THE CONVERGENCE OF ENVIRONMENTAL INFLUENCES AS POTENTIAL PRECIPITATING FACTORS OF AML-M2

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ABSTRACT

Acute myeloblastic leukemia is known as an insidious, often times fatal, disease; however, its etiology has not been fully elucidated. This work was conducted so as to explore the potential environmental influences that may converge and precipitate a myelodysplastic event or even a leukemic disease state. Environmental chemicals were the primary focus of this investigation, including: the fertility drug clomiphene citrate, (Clomid); and a combination of pesticides commonly applied to produce. Water samples were extracted from Berlin Lake, as well, to gauge recreational water contamination. The Berlin Lake water samples were found to contain a number of hydrocarbon contaminants; with the main supplier of such contaminants believed to be the 'fuel-dumping' of recreational crafts.

STATEMENT OF PROBLEM

This thesis attempts to marry two not unlike disciplines, pathology and environmental chemistry; in that this work explores the hematology/pathophysiology of acute myeloblastic leukemia (M2)-in relation to this disease state's etiology. And it is within the etiology of AML that this effort becomes totally intercalated with environmental chemistry. Environmental chemistry, the term itself, quite often evokes ideas or images of chemical pollutants-what are they? Where are they? What chemistry/reactions are they capable of?-an area of chemistry which is, by many, never actually thought of in tandem with the medical arts. For if it were, perhaps questions such as: What is the minimum lifetime level of exposure for that compound? What chromosomal aberrations are associated with such an exposure? Are there consequences to in utero exposure? Will these compounds ever safely degrade? Would be more closely associated with this discipline. It is just such questions that will be brought to the fore in this thesis, regarding AML. The author would like to not only introduce AML as a possible "environmental disease", but to also present a somewhat rudimentary case study of a nine year old boy recently diagnosed with AML M2; a diagnosis which was critically influenced by the child's annual exposure to a contaminated water source.

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Chapter 1: How Blood Cells are Produced

Hematopoietic Tissue

Blood cell production occurs at various points of or within the human body during the course of development from embryonic through adult life. Formation of the blood cells first begins within the yolk sac of the embryo; later shifting to the liver and to a lesser extent the spleen-allowing these organs to then become the dominant production sites between the second and seventh months of gestation. The liver and spleen are then superceded by the bone marrow-which then serves as the main and most important site of blood cell production *post-partem*. Lymphocyte production is the only exception to this; for their production occurs to a greater extent in the lymphoid tissues *post-partem*. Hemapoietic tissue occupies all of the cavities within the bones of the neonate; with subsequent corporeal development however, this tissue becomes localised in the cavities of the upper shafts of the femur, and humerus, the pelvis, spine, skull and bones of the thorax. The total volume of hematopoietic tissue within the adult body is between one and two liters. It is referred to as red marrow primarily due to its macroscopic presentation; the bone marrow within the more peripheral regions of the body/skeleton is predominantly composed of adipose cells/adipocytes and is known as yellow marrow. Yellow marrow claims a volume of between one and two liters as well; serving as a reserve space into which hematopoietic tissue

can expand if, for example, the body should suffer an increased need for blood cells and their production. Extramedullary hemapoiesis or hemopoietic activity exclusive to the liver and spleen occurs in adult life only in rather rare pathological conditions.[1]

Bone Marrow Structure

The red marrow, as found between or intermingled with the trabeculae of bone within the marrow cavitiy, proper, houses specialized connective tissue cells: reticulin fibrils, blood vessels, fat cells, nerves and macrophages-along with cells of the lymphoid and hemopoietic series. Fine reticulin fibrils help to create a supportive framework for the bone marrow components. These fibrils extend/reach from the endosteum of the bony trabeculae to the vascular sinusoids and are believed to be produced by the adventitial reticular cell.

Adventitial Reticular Cell

The abluminal or adventitial surface of the marrow's vascular sinus consists of reticular cells. These cell bodies are contiguous with the vascular sinus, thereby contributing to a portion of its adventitial coat. The adventitial reticular cell possesses extensively branched cytoplasmic processes which enwrap the outer wall of the marrow's sinus-forming an adventitial sheath.[2]

The reticular cells synthesize argentophilic fibers that, in conjunction with their cytoplasmic processes, reach into the hemopoietic recesses of

the/ within the marrow; these fibers help to construct a framework upon which hemopoietic cells rest. The cell bodies, their broad processes and fibers help to compose the reticulum of the marrow.[2] The membranes of the adventitial reticular cells are known to contain high levels of alkaline phosphatase; express CD10, CD13 and class 1 HLA antigens; are positive for the 6/19 antibody; express nerve growth factor receptors; differentiate along the smooth muscle pathway; and contain alpha smooth muscle actin, vimentin, laminin, fibronectin and collagens 1, 3 and 4. These reticular cells are commonly CD34 antigen-negative.[2]

Fibronectin

Fibronectin is known to localize at the sites of hemopoietic cell and marrow stromal cell attachment. Early erythroid progenitor cells attach themselves to the cell-binding domain of fibronectin. Additionally, adhesion of granulocyte hemopoietic cells to stroma is mediated for the most part by fibronectin. Such a binding can be strengthened via protein kinase C activators-phorbol esters, for example-thereby suggesting the possible involvement of integrin receptors in the cell-attachment process.[2]

Collagen

Collagen types 1 and 3 are produced by fibroblasts within the marrow and are associated with microvascular walls; type 4, however, is confined to endothelial-type cells and their basal lamina. Marrow derived fibroblasts along with stromal cells, synthesize collagens 1, 3, 4, 5, and 6.[2]

Laminin

Laminin is a multidomain glycoprotein with both mitogenic and adhesive sites; it is a main component of the marrow extracellular matrix and basement membranes. This glycoprotein reacts with collagen type 4 and assorted proteoglycans to regulate leukocyte chemotaxis. In a similar manner, CD34 positive granulocytic progenitors, mature monocytes, and neutrophils attach to laminin. Laminin is believed to have a part in strengthening adhesive interactions with integrin receptors, specifically receptors $\alpha 5\beta 1$ and $\alpha 6\beta 1$-on the surface of hemopoietic cells within the cytomatrix.[2]

Thus, it becomes apparent that marrow structure is critical for proper hemopoietic activity; for it essentially provides a framework-as generated by the adventitial reticular cell population, reinforced by the likes of fibronectin, collagen and laminin-upon which or within which the hemopoietic cell hierarchy is able to attach and differentiate. It is with this structure then that the majority of the blood cell population, within the general circulation of the body, is maintained.

With this juncture in the discussion, it becomes critical to recognize and/or accept several generalisations concerning bone marrow, before hemopoiesis-as involving blood cell production and differentiation-is able to be considered. These generalisations are as follows:

1. in marrow there exists a hierarchy of hemopoietic cells;

with the primary or initial cell being referred to as the multipotential stem cell;

2. stem cell differentiation is unidirectional and is closely aligned with the restriction of any cell renewal capacity;
3. proliferation of the stem cells is wholly dependent upon contact with the marrow's stromal cells;
4. the total overall proliferation and differentiation of stem cells is regulated by local and systemic growth factors and their accompanying inhibitors.[3]

Hemopoiesis

It is universally accepted that blood cells develop from a small population of stem cells or progenitor cells within marrow; which maintain their population via self-replication and also give rise to precursors of various other blood cells.[1] The progenitor cells spend the majority of their existence in an out-of-cycle Go phase; during this phase of the cell cycle they are preoccupied with DNA repair and other forms of genetic maintenance. Throughout the duration of this rather quiescent phase, the cells are less susceptible to genetic damage by ionizing radiation, alkylators, and viruses. As the number of cells reaching the terminus of the G1 phase increases-stem cells are prepared to react within an approximate thirty minute window to an array of stimuli; either carried to them via the general circulation or as produced directly in the marrow.[3]

Following this post-G1 accumulation and subsequent cellular activation towards differentiation, proliferative activity progressively increases. It is at this post G1 accumulation that the blood cells are said to be in the maturation compartment; in which a cascade of morphological changes occurs without cell division-to yield the mature end-cell. The range of different blood cell series which can develop from a particular precursor progressively declines as the precursor comes to possess an increased degree of differentiation.[1]

The Stem Cell

The stem cell or hemopoietic stem cell is often considered as an example of the most primitive cell type. This fundamental cell is able to divide; however a subset of the stem cell population will remain unmodified during the maturation or stem cell phase. The cells that remain in this "un-specified" state are/compose the pluripotential cell population.[4]

A small number of the cells in the maturation or stem cell compartment are forever undergoing mitosis; this constant cell-set is responsible for maintaining a relatively homeostatic blood cell population. For example, when the demand for blood cells is intensified, the percentage of dividing stem cells-both uncommitted and committed-increases. This stimulus to differentiate for committed cells of each cell line, is mediated by glycoprotein inducers or hemopoietins; including erythropoietin, thrombopoietin and assorted leukopoietins.[4]

It is this complex cascade of hemopoietic events, as occurring within the bone marrow, that produces the blood cells that sustain mammalian life. Upon closer examination, however, there becomes apparent an intrinsic fragility to this system of red blood cell (RBC), platelet, and white blood cell (WBC) production; these cells-to be identified in the chapter forthcoming-are not only at the very foundation of life, but, as it will be shown, are also the first to fall victim to mutation and disease.

Chapter 2: Description and Function of Blood Cells

Red Blood Cells

The mature red blood cell is a rather unique development in cellular evolution; for it has developed so as to exclude all biosynthetic organelles such as nucleus, ribosomes and mitochondria. Essentially, the rbc has become a sort of hematologic minimalist, in that it possesses just enough, biochemically speaking, to adequately fulfill its role of oxygen deposition and carbon dioxide removal within the body. The rbc has developed into a rather flexible biconcave torus shape-brilliantly formed so as to maneuver through the blood vessels composing the body's microcirculation. The mature rbc will travel in upwards of 1 million times through the body equaling a distance of about 300 miles. A normal, mature red blood cell will measure approximately 7.8 μm in diameter, 1-7 μm in width and have a volume of 94 ± 14 fL and a surface membrane area of 135 ± 16 sq μm. Such a surface area enables these cells to not only endure the hydraulic bending forces of non-laminar circulation, but adjust to various instantaneous contortions without damage or retardation of progress. Additionally, the biconcave shape of these corpuscles allows for a quite favorable surface area to volume ratio; thereby allowing them to travel across and/or through cylindrical capillaries only 5 μm in diameter, via the adoption of an umbrella shape transverse to the direction of blood flow.[3]

Membrane Properties

The structural elements of the red blood cell membrane that make the aforementioned progress possible include:

1. a lipid bilayer, composed of phospholipids and non-esterified cholesterols; providing a semipermeable barrier between the internal cell cytosol environment and the external environment of the blood stream, proper;
2. transmembrane proteins;
3. a membrane skeleton that essentially sheathes the internal or cytosolic side of the cell-affording it (the cell) a high degree of structural stability or integrity.[2]

Composition

The vast majority of the membrane's phospholipids are phosphoglycerides-consisting of a glycerol backbone; the hydrophobic long-chain fatty acids are anchored to glycerol's first two carbons. The residues, which determine phospholipid specificity are linked to the third carbon of glycerol by means of phosphoester linkages and are exposed at one of the lipid bilayer surfaces. The involved phosphoglycerides are of the following mix: 27% of the total phospholipids-phosphatidylethanolamine; 28%-phosphatidylcholine; 13%-phosphatidylserine, along with phosphatidylinositol. Sphingomyelin constitutes the other phospholipid contributor; it consists of a hydrophilic moiety identical to that of

phosphatidylcholine; however, the hydrophobic region is composed of ceramide. It is important to note that ceramide contains sphingosine along with a fatty-acyl side chain attached to sphingosine's amino group. In addition, cholesterol fits into the membrane in its unesterified form.[2]

Cell Surface

The surface of the rbc is fortified via neuraminic acid residues, which impart a negative surface charge to the cell. Any deviation in cell surface charge is anything but salubrious in regards to the health of the erythrocyte. The red blood cell surface antigens are found on the glycolipids; *ie* the glycosphingolipids or upon the externally exposed portions of transmembrane proteins or their carbohydrate side chains.[2]

Membrane Permeability

The normal erythrocyte membrane is virtually impermeable to monovalent and divalent cations. This helps to maintain a high potassium, low sodium, low calcium cellular content. Anions, however, are exchanged via the anion transport protein. The rbc cell membrane is also known to contain at least one water channel protein that facilitates the rapid movement of water molecules across the membrane; because of these channels, the erythrocyte behaves as a perfectly, or very nearly so, run osmometer. Glucose is carried via a glucose transporter protein, while larger, charged molecules do not travel across the rbc cell membrane.[2]

Red Blood Cell Function

The ultimate design of the erythrocyte is the ability to transport oxygen and carbon dioxide, the respiratory gases. Hemoglobin picks up oxygen in the pulmonary capillaries and delivers it, via the rbc, to tissue capillaries; within the tissues, oxygen is exchanged for carbon dioxide-a byproduct of cellular metabolism. A human, at rest, consumes approximately 250mL of oxygen and exhales around 200mL of carbon dioxide, every minute. Dissolved as a gas in plasma water, only about 5mL of oxygen can be delivered to needy tissues each minute. Whole blood is capable of delivering 200 ml of oxygen/liter, due primarily to red cell hemoglobin, to tissues within the body. For this oxygen delivery to occur, hemoglobin must bind oxygen with an intensity that allows it to be removed from pulmonary capillaries at high oxygen tensions; while still being able to deliver/unload oxygen at the decrease oxygen pressure of the tissues; hemoglobin must meet/attach to oxygen with flawless affinity.[3]

Platelets

Platelets are formed in the Golgi region of the cytoplasm of megakaryocytes and are released into the blood via cytoplasmic fragmentation. Thrombocytopoiesis is under the dictate of thrombopoietin. Although the majority of the blood's platelets are produced by megakaryocytes within the bone marrow, a small number is believed to be

derived from pulmonary megakaryocytes.[4] Platelets store a number of molecules that influence platelet function, vascular tone, fibrinolysis and wound healing; these compounds are released upon platelet activation.[2]

The mean diameter of platelets is variable; generally the platelet is between 1.5 and 2.5 μm across, only about 1/3 to 1/4 the diameter of rbc's. Platelets have even been observed to possess filopodia-or long, thin processes extending outward from the platelet body proper.[2] Platelets possess a glycocalix, which extends 14 to 20 μm from the cell surface. This 'coat' is believed to consist of membrane glycoproteins, glycolipids, mucopolysaccharides and adsorbed plasma proteins. The platelet surface is host to a network of indentations, thought to represent openings of the cell's open canalicular system, a complex channel system that communicates through out the cell. In addition, platelets, in an electric field, react or move as is they were influenced by a net negative surface charge. This net negative surface charge is created, in part, by sialic acid residues attached to proteins and lipids along the cell-surface. The overall electrostatic repulsion created via this charge, is believed to aide in the prevention of at-rest platelets from adhering to others or the negatively charged cells of the endothelium.[2]

Membrane Properties

The plasma membrane is a trilaminar unit consisting of a bilayer of phospholipids within which is embedded cholesterol, glycolipids and assorted glycoproteins. This membrane is believed to house the Na^{+} and Ca^{+2} ATPase pumps, which are integral in controlling the platelets' ionic environment. The phospholipids, which help to create and stabilize the plasma membrane, are distributed in a rather asymmetrical pattern; with those negatively charged phospholipids, phosphotidylserine in particular, are known to accelerate the coagulation cascade. Additionally, select membrane phospholipids are enriched with arachidonic acid; thereby providing a repository of arachidonic acid ready for release and subsequent conversion into thromboxaneA2, often referred to as TXA2.[2]

Organelles

The sol-gel zone or platelet interior, houses two types of granules: the α granule and the dense bodies; along with sparse mitochondria and glycogen deposits. The α granules outnumber the dense bodies within the platelet; are contained by a membrane and hold hydrolytic enzymes- including acid phosphatase, β-glucuronidase, and cathepsin; the dense bodies are enriched in serotonin and derived from α-granules.[4] They also contain ATP and ADP in a 2:3 ratio respectively. Storage of these adenine nucleotides is believed to be done via a vertical stacking of the molecules' rings. The planar hydroxyindole rings of serotonin may also aide in the

construction of these aggregates.[4] Decrease of the contents of the dense granules, from activated platelets, is part of a fundamental positive feedback reaction for platelet aggregation; based upon ADP being a rather strong platelet agonist and serotonin a weak agonist.[4]

Function

The platelet, in response to strong activators, such as: adhesion to exposed collagen with blood vessels following a vascular trauma; adhesion to atherosclerotic blood vessel walls following plaque rupture; thrombin and/or elevated collagen concentrations[2]; undergoes a biochemically prescribed sequence of events. This sequence includes: a distortion of shape; adhesiveness; primary aggregation; secondary aggregation; and release reactions. The sequence realizes completion if the inducer is requisitely strong with no accompanying inhibitors. If the inducer should be weak, however, with subsequent activation of one or more inhibitors, then the response can stop and actually reverse. The inducer also helps to predict whether the response sequence is followed in its entirety. This hemostatic process is inclusive of/to the activation of the blood coagulation response, as well.[4]

White Blood Cells

Classification of Lymphocytes

Mature lymphocytes, although originating from a common parent cell, are divided into several functional types. These functional types include: T-cells, B cells and the intriguingly titled natural killer (NK) cells; however, the scope of this discussion will be narrowed to include only T and B-cells.[2]

T-cells

T-lymphocytes are the predominant lymphocyte in blood and lymph. In the lymph nodes, T-lymphocytes are known to localize within the dense corona of lymphoid follicles in addition to congregating in the interfollicular and subfollicular zones; within the spleen, they are found within the outer mantle of the periarteriolar sheath. When sensitized T-cells are activated, they produce lymphokines; furthermore, upon activation, T-cells are able to behave as effectors, helpers and/or suppressors. Effector T-cells are integral in the delayed hypersensitivity reaction along with the graft-verus-host reaction; whereas helper T-cells promote or enhance antibody production by B-lymphocytes; and suppressor T-cells inhibit antibody production by B-cells.[4]

T-cells possess CD2, CD4 and CD8 receptors; these receptors are simply adhesion molecules and/or signal transducers. The CD2, CD4 and CD8 receptors react with a number of cell surface ligands, including the lymphocyte function-associated antigens (LFAs), LF1 and LF3. As an example of this relationship, consider the following: the conjugation of CD2 to LF3 ligands promotes nonspecific adhesion of T-cells to antigen

presenting cells (APCs) and by doing so, facilitates antigen recognition and T-cell activation. In complement, the interplay between CD4 or CD8 receptors with MHC (major histocompatibility complex) proteins reinforces the bond strength of specific T-cells to APCs. Following this contact, T-cell recognition by the T-cell antigen receptor (TCR) and APC binding through MHC complementarity induces lymphocyte effector function; for example, cytolysis, or lymphokine production. Following this biochemical crescendoing, the T-cell involved, disengages from the APC, creating a vacant site for the attachment of additional antigen-specific resting T-cells.[3]

B-cells

B-cells are relatively short-lived, and originate from within the bone marrow. They have a number of surface immunoglobin receptors, (IgM, IgA, IgD. IgG, IgE), and fundamental in the construction and maintenance of the humoral defense system.[4] B-cells have developed so as to be able to recognize a seemingly infinitesimal number of potential antigens; and following contact with the antigen, they convert to plasma cells; with the function of the plasma cell ultimately being antigen-secretion. When a B-cell is activated, via antigen contact with an Ig receptor, a clone of antibody-secreting plasma cells is produced. A minority of the activated cells divide only briefly; however, they will survive as 'long-lived memory cells'.[3]

Chapter 3: Acute Myelogenous Leukemia (AML)

Pathophysiology of AML

In prefacing the definition of AML, acute myeloid leukemia, it is necessary to define the cancer, leukemia. Leukemia is a malignancy of the blood-forming cells; occurring when immature or mature cells multiply uncontrollably within the bone marrow. This condition is identified as lymphocytic or myeloid depending upon which cell-line is altered. Leukemia is then considered to be acute or chronic; acute leukemia being characterized by a disease in rapid progression with a predominance of blastic or highly immature cells; whereas chronic leukemia signals a disease developing at a much slower rate with an increased number of mature cells.[1] More specifically then, acute myeloid leukemia is identified as the rapidly progressing neoplastic growth of immature myeloid cells or myeloblasts; and because the nonlymphoid cell-lines are involved, this malignancy is equally recognized as acute nonlymphocytic leukemia or ANLL (ACS). Briefly then, the myeloid cell lines include the following sequences of cell maturation within the bone marrow:

*stem cell → myeloid stem cell → erythroblast → reticulocyte → erythrocyte

*stem cell → myeloid stem cell → megakaryoblast → promegakaryocyte → megakaryocyte → thrombocyte

*stem cell → myeloid stem cell → myeloblast → basophil

*stem cell → myeloid stem cell → myeloblast → eosinophil

*stem cell → myeloid stem cell → myeloblast → neutrophil

*stem cell → myeloid stem cell → monoblast → promonocyte → monocyte → macrophage.[2]

Thus, surveying the number of rather complicated cell-differentiation schemes, it becomes increasingly clear that there exists a rather extensive list of site combinations where a perversion of cell-development can occur. For example, a derangement in the myeloid cell line at a pluripotential/stem cell, gives rise to a disease sequelae of vastly different dynamics than would a derangement occurring further along the differentiation scheme.[3] However, any sort of renegade or neoplastic proliferation of cells such as myeloblasts, erythroblasts and the like, not only encourages genetic misprints in these unchecked cells-but by the sheer numbers of cells being 'over-produced'-healthy marrow cells are dislodged. This usurping of cell position, within the marrow matrix, manifests itself within the patient as anemia, neutropenia and thrombocytopenia.[2]

Anemia-a reduction in the quantity of the oxygen-carrying pigment or hemoglobin, within the blood; the main symptoms include: excessive tiredness and fatigability, breathlessness on exertion and poor resistance to infection.

Neutropenia-a decrease in the number of neutrophils in the blood; resulting in an increased susceptibility to infections.

Neutrophils-a variety of white blood cell, distinguished by a lobed nucleus and the presence in its cytoplasm of fine granules that stain purple with Romanovsky stains. The neutrophil is capable of phagocytizing bacteria and contributes to the body's defense against infection

Romanovsky stains-a group of stains used in the microscopic examination of blood cells, consisting of variable mixtures of thiazine dyes; such as azure B with eosin. Romanovsky stains communicate characteristic patterns, on the basis of which blood cells are classified. This group of stains includes stains of: Leishmann, Wright, May-Grunwald, and Giemsa.

Thrombocytopenia-a reduction in the number of platelets within the blood. This condition results in bleeding into the skin, spontaneous bruising, and prolonged bleeding after injury. Thrombocytopenia may result from failure of platelet production or their excessive

destruction.[5]

Clinical Presentation of AML

The general clinical signals that may indicate the development of AML, within a patient, include: pallor, fatigue, weakness, palpitations and dyspnea (heavy or laboured breathing) upon exertion-all being symptoms which communicate anemia. Whereas, easy bruising, petechiae (red skin spots signifying bleeding into the skin), epistaxis (nosebleed), gingival bleeding, conjunctional hemorrhages and prolonged bleeding following superficial skin injuries, are symptoms characteristic of thrombocytopenia. In addition, fever is present in the majority of patients upon diagnosis; as is palpable splenomegaly and/or hepatomegaly.[1]

Although there are many combinations of/profiles of symptoms at diagnosis, perhaps the most accurate and fail-safe infrastructure to construct a diagnosis around is the hematological findings/CBC numbers and the initial personality of the bone marrow.

A bone marrow biopsy of a patient suspected of developing AML, will always contain leukemic blast cells.[2] Myeloblasts are identified within the biopsy via three pathognomonic features: reactivity with a series of histochemical stains; the presence of Auer rods within the cells; and/or reactivity with specific monoclonal antibodies against epitopes found on the surface of the myeloblasts.[2] Additionally, normal erythropoiesis,

megakaryocytopoiesis and granulopoiesis are significantly reduced or non-existent within the biopsy. The aspirate of marrow may also contain isolated clusters of erythroblasts or megakaryocytes.[2]

The blood values of a patient suspected of developing AML-often communicate a suspicious WBC level; many times being either superelevated or subnormal. The myeloblast population within the blood is not necessarily valuable in determining the extent of leukemic cell infiltration into the body, but is valuable in gauging disease progression. Blast counts in excess of 100,000 cells/μL indicate the potential for a terminal progression and scream of the risk of the formation of 'leuko-occlusions' within blood vessels of the lungs and brain. Such CNS vessel occlusion, by gummy accretions of sticky myeloblasts, contributes, if not, precipitates, life-threatening neurological damage; for example, fatal cerebral hemorrhage.[3]

Types of AML

Acute cases of myelogenous leukemia are classified according to the French-American-British (FAB) identification scheme. The type of AML is assigned a label of M1-M7; accompanying each designation is a set of hematological thresholds that must be met to allow for such a diagnosis. The categories and hematologic criteria conform to the following:

AML M1-Myeloblastic leukemia: At least 30% of the nonerythroid cells within the marrow are recognized as myeloblasts;

with a minimum of 3% of blasts staining for myeloperoxidase or granule phospholipid via treatment with Sudan Black.

AML M2-Myeloblastic leukemia with maturation: At least 30% of the nonerythroid marrow cells are myeloblasts; promyelocytes account for more than 10% of the population and monocytic elements more than 20%.

AML M3-Promyelocytic leukemia (APL): The majority of marrow cells are abnormal hypergranular promyelocytes; Auer rods may be present within a small percentage of these promyelocytes.

AML M4-Myelomonocytic leukemia (AMML): At least 30% of the nucleated marrow cell population are blasts; with granulocytics accounting for more than 20% of the nonerythroid marrow cells.

AML M5-Monocytic leukemia (AMoL): At least 30% of the entire nonerythroid marrow cell population are monoblasts, promonocytes or monocytes. In the M5A subtype, a minimum of 80% of all monocytic cells are monoblasts.

AML M6-Erythroleukemia (AEL): A minimum of 50% of the nucleated marrow cell population are erythroid precursor cells; in addition, at least 30% of the remainder of nonerythroid cells are blasts.

AML M7-Megakaryoblastic leukemia (AMegL): The marrow biopsy displays at least 30% of the cells present to be of megakaryocytic lineage.[3]

As mentioned within the preface, a specific case of pediatric AML M2-myeloblastic leukemia with maturation-will be the primary focus of this discussion.

Chapter 4: The Pharmaceutical Suspects
What was the precipitating factor/s to the development of AML in this case study?

As spoken to within the preface-this thesis, ultimately, is an expanded case study; the focus being a then nine year old male, diagnosed with AML M2. Having characterized the disease state in chapters preceding, the question of HOW? arises. More precisely, how exactly is a child at risk for developing leukemia-myelogenous leukemia at that? The first possibility to be explored is maternal/paternal chemical exposure, followed by infant chemical exposure.

When analysing the chemical exposure of the diagnosed young man and his parents, it becomes obvious, quite rapidly, that two pharmaceutical agents are of paramount importance to this discussion; sulfisoxazole and clomid (clomiphene citrate). Sulfisoxazole is somewhat suspect in that, as an infant, this young man received 18gm, in <5 days, as treatment for otitis media; 18gm, in <5 days, in a pediatric context, is considered an overdose.[6] Of equal, if not greater, suspicion is clomiphene citrate or clomid, a fertility drug taken by the mother, to induce ovulation. Clomid regimens, as will be presented, are notorious in their ability to encourage fetal/neonatal structural malformations; in conjunction with chromosomal abnormalities and leukemia within the neonate.[7]

Clomid

Clomid, is identified by its manufacturer, Hoechst, as an orally administered, nonsteroidal, ovulatory stimulant; chemically identified as 2-[p-2(chloro-1,2-diphenylvinyl-phenoxy]triethylamine citrate. Clomid or clomiphene citrate, is a mixture of two geometric isomers, [cis(zuclomiphene) and trans(enclomiphene)] containing from 30-50% of the cis-isomer.[7]

Clomid has the potential to interact with those tissues rich in estrogen-receptors; these tissues include, but are not limited to, the hypothalamus, pituitary, ovary and endometrium. This drug may also compete with estrogen for estrogen-receptor binding sites and may retard the renewal of intracellular estrogen-receptors. Essentially, clomid initiates an endocrine cascade ending in a preovulatory gonadotropin surge, pre-empting follicular rupture. The first event in this cascade is a marked increase in the release of pituitary gonadotropins. This increase encourages steroidogenesis and folliculogenesis; thereby promoting the growth of the ovarian follicle and increases in circulating estradiol levels.[7]

Although such an endocrine cascade may be the exact desired result-a number of the risks and contraindications may unfavourably skew the benefit/risk ratio of clomid therapy. The outward or obvious upon parturition, malformations which are experienced by the subject of this study, include: undescended testicles, inguinal hernia and umbilical hernia.

All three of these structural abnormalities are specifically cited within the physician's package insert for clomid, as possible risks. In addition, the risk of neoplasms and chromosomal disorders are cited: the neoplasms listed directly include neuroectodermal tumour, thyroid tumour, hepatoblastoma, and most importantly for this discussion, leukemia. It is also worthy of mention, that clomiphene citrate is contraindicated for women known to suffer from organic intracranial lesions-such as pituitary tumour.[7] In the instance of the nine year old subject, his mother has been diagnosed with just such an intracranial lesion, a pituitary tumour; her diagnosis preceded her clomid therapy.

In addition to the mother's anovulatory condition, the child's father was identified as suffering from oligospermia. The parameters for oligospermia are between 0.5-20 million sperm/ml[8]; with normal serum gonadotropins and testosterone. However, although clomid/clomiphene may be taken at doses up to 100mg/day, in treatment of male infertility, due to a miscommunication, the child's father took two times the prescribed dose during treatment. It has been suggested that extremely high or low concentrations of clomid/clomiphene negatively impact both sperm motility and fertilising capacity.[9] Please consider the risks that begin to intensify for the fetus-considering that both parents are

receiving clomiphene therapy and one is receiving two times the prescribed dosage. If there was "acceptable risk" with exclusively mother receiving fertility therapy, did the line between benefit and risk become a bit muddled when father began treatment, and two times the treatment at that?

Sulfisoxazole

Sulfonamides

Sulfonamides, the general category of pharmaceuticals to which sulfisoxazole belongs, are synthetic derivations of p-aminobenzenesulfonamide. A benzene ring with a sulfonamide group and a primary amino group *para* to the sulfur side-chain, impart antibacterial activity to the compounds. Substitution of the N^4-amino group with groups e.g. radicals, that may be easily converted to a free amino group within the body, allow the compound to retain antibacterial activity. Furthermore, any substitutions within the N^1-amide group produce compounds different in solubility, protein binding, tissue distribution, and rates of metabolism and excretion. The most effective sulfonamides are those obtained via substitution of heterocyclic groups in the N^1 position.[6]

Sulfonamides are principally bacteriostatic; in that they directly disrupt bacterial utilization of p-aminobenzoic acid (PABA) within the biosynthesis of tetrahydrofolic acid cofactors. This interference is possibly due to sulfonamides being structural analogs of p-aminobenzoic acid; thereby

being capable of competitively inhibiting dihydropterate synthase. Dihydropterate synthase catalyses dihydropteric acid formation from PABA and pteridine. Dihydropteric acid is a tetrahydrofolic acid precursor. The bacteriostatic potential of the sulfonamides is only realised against microbes that synthesize their own folic acid.[6] Thus sulfonamides are effective against gram positive bacteria, including: strains of *Staphylococci, Streptococci, Bacillus anthracis, Clostridium tetani, Clostridium perfringens,* along with a number of strains of *Nocardia asteroides* and *Nocardia brasiliensis.* The gram negatives which thay are effective against include: *Enterobacter, Escherichia coli, Klebsiella, Proteus mirabilis, Proteus vulgaris, Salmonella* and *Shigella.*[6]

Sulfisoxazole

Sulfisoxazole, the sulfonamide specific to this discussion, shares the actions and uses of the sulfonamides. However, there exist a number of adverse reactions with this sulfa drug, as with many others. The most pertinent, here, being the sulfonamide-induced blood dyscrasias: agranulocytosis, hemolytic, aplastic or megaloblastic anemia, leukopenia, thrombocytopenia and eosinophilia.[10] The blood dyscrasias are believed to be provoked by both an immunologic reaction, involving haptene formation and destruction by antibodies, and an idiosyncratic mechanism. It has been demonstrated that such toxic effects, from sulfa drug therapy, occur after a latent or window period, anywhere from 2 to 36 months, following

treatment.[11] Please consider the course of sulfa drug treatment taken by the subject: 18gm, <5 days, powder form, in a pediatric context; it is of vital importance, when analysing this therapy, to recognise that not only does this scenario constitute an overdose-but the sulfisoxazole was not prepared in the pharmacy, as the prescribed suspension. The prescription was however, filled and given to the patient's parents with minimal instruction; not enough instruction to allow them to realise that sulfisoxazole is not/should not be administered as a powder-to be sprinkled over a patient's cereal! Such miscommunication and negligence could only endager this child, and most probably set him up for an increasing susceptibilty to a hematologic event, such as leukemogenesis!

Chapter 5: The Environmental Suspects

Environmental chemical exposure of both parents and child is perhaps best divided into two distinct groups of chemicals: Berlin Lake water contaminants and fruit and vegetable contaminants. The Mahoning River Basin plays a key role in this case study due to the child's repeated annual exposure to Berlin Lake water via swimming, diving and boating. Whereas preservative chemicals, common to fruits and vegetables are integral in that the child's father has worked in the produce department of a local grocery chain for nearly thirty years.

Berlin Lake

Appendix C, of the EPA's May 1, 1996 report on the Mahoning River Basin, catalogues over 495 known spills into the basin and its tributaries, between 1983-1994. Before discussing some of the more serious spills, it is essential to note that the level of sophistication or better yet, exactitude in identifying what exactly spilled and in what volume, is of an incredibly-even frightfully low level. For example, entries of spills of "waste water-quantity unknown" or "sewage-quantity unknown" or "unknown white stuff-quantity unknown" or "suspended solids, yellow material, orange stuff, illegal dumping, junk/trash"-all recorded "quantity unknown"-are representative of how the government has documented spills into a waterway, known of and used in almost exclusively a recreational capacity.

Some chemicals that have been recorded as spilling into this waterway in excess of thousands of gallons, include: 1,3-butadiene, assorted fuel oils, asbestos, ethylene glycol, propylene glycol, and 2,4,6-trinitrotoluene.[12]

The first, most obvious and unfortunately the most frequently spilled chemicals, are those, as previously mentioned, which belong under the heading of hydrocarbons or petroleum distillates. The petroleum distillates have increased toxic effects when they are aspirated into the tracheobronchial tree than when they are ingested; ingestion of between 500-1000mL may cause minor symptoms, whereas aspiration of just 1mL can lead to lethal chemical pneumonitis.[8] Pesticides, camphor, halogenated compounds and metals, if dissolved in petroleum distillates, can significantly increase this toxicity.[8]

Petroleum distillates are recognised as fat solvents, capable of altering nerve function, potentially leading to depression, coma and convulsions. Benzene contaminants of the distillates, may potentiate adverse effects on liver, kidney and bone marrow function.[13] Laboratory findings, based upon exposed individuals, tell of reduced RBC counts, bone marrow hypoplasia and the presence of protein and RBC's in the urine.[13]

1,3-Butadiene

1,3-Butadiene is produced during petroleum processing. It is the 36th highest volume chemical produced in this country. 1,3-Butadiene is

recognised by the DHSS as a human carcinogen. Exposure to this compound is possible via: urban or suburban air in or around chemical, plastic or rubber facilities; air contaminated from car/truck exhaust or waste incineration; cigarette smoke; drinking/swimming in water near production or waste sites; and skin contact with gasoline.[14] The occupational exposure limit, as established by OSHA, for 1,3-buatdiene, is 1000ppm of air.[15]

Fuel Oils

Fuel oils are, obviously, a veritable hydrocarbon cocktail, produced directly from crude oil petroleum; and include kerosene, diesel fuel, jet fuel, range oil and home heating oil. Fuel oils, when spilled into water are not degraded into more benign or eco-friendly compounds, quite the contrary. Rather, these petroleum by-products may dissolve in water and/or eventually be deposited in the waterway's sediment. Furthermore, fuel oils, of any sort, are recognised as bioaccumulators-simply meaning that they accumulate in the adipose of any exposed creature-fish, bird, human, or otherwise. One of the most direct routes of exposure is the immersion or consumption into/of contaminated water. Unfortunately, prolonged and/or repeated exposure to such compounds adversely effects kidney function and interrupts the prothrombin and fibrin sequences within the blood; thereby significantly increasing clotting times.[14]

Used Mineral-Based Crankcase Oil

This compound differs from unused oil in that it contains additional chemicals formed via high temperature and high pressure exposure within an engine. It also contains an assortment of metals from engine parts, in addition to gasoline, antifreeze and byproducts of spent gasoline.[14] When such a mixture invades the environment, it acts in much the same way as fuel oils. They find their way through waterways, accumulating in low-layer sediments, animals, fish and humans. Therefore, exposure to contaminated water or soil would be possible delivery routes into the body. And once an individual has suffered such a repeated exposure, hematological events, such as anemia, become increasingly likely to occur. In addition, used oil contains PAH's or polyaromatic hydrocarbons-which are recognised carcinogens.[14]

Contaminants, other than the petroleum distillates, which have been spilled into the Mahoning River Basin/Berlin Lake, include: asbestos, ethylene glycol, propylene glycol and 2,4,6-trinitrotoluene.[12]

Asbestos

The term asbestos is applied to any mineral that decomposes into fibres. Chrysotile, the most common form, is fibrous serpentine, a magnesium silicate containing 40% silica. Its fibres are tubular in cross-section and as small as 0.015μm in diameter. Crocidolite, another form, is fibrous riebeckite, a sodium ferro-ferrosilicate, which is 41% silica. Its

fibres can be as minute as 0.08μm in diameter. A third form, amosite, is fibrous grunerite, a magnesium ferrosilicate, 49% silica. Amosite fibres are as little as 0.1μm in diameter. Asbestos also includes anthophyllite and termolite-actinolite. Uses for the various forms of asbestos include: cloth production, brake linings, cement products, paper, flooring, gaskets and paint; a total of 3 million tonnes is produced annually in the United States.[16] Asbestos does not readily degrade within the environment, it merely settles-in water, soil, and within animals. Asbestos is capable of bioaccumulation. Inhalation of asbestos fibres increases the risk for lung cancer and mesothelioma, which is a cancer of the pleural membrane. Whereas, ingestion of such contaminated water, has been shown to elevate the risk for stomach, intestinal, esophageal, pancreatic and kidney cancers.[17] The EPA has set a limit of 7 million fibres/L as the highest concentration of long fibres acceptable within drinking water.[14]

Ethylene and Propylene Glycol

Ethylene glycol and propylene glycol are clear, colourless, liquids-best described as 'syrupy' at room temperature. Both glycols are main components of anti-freeze and de-icing solutions for cars, boats and airplanes; and are used in the manufacturing of polyesters-also as solvents in the plastic and paint industries.[12]

The fatal dose of ethylene glycol is approximately 100g. Whereas the exposure limit for particulate ethylene glycol is 10mg/m^3; 50ppm for

vapour.[18] Ethylene glycol and its esters are distributed with metabolic water and are metabolised to oxalic acid within the body; it is this conversion that is believed to be involved in some of its toxic effects.[19] The ethers of ethylene glycol, although not degraded to oxalic acid, idiopathically produce brain and kidney damage.[18] The majority of the glycols produce profound acidosis.[19]

The pathology of a glycol poisoning may include congestion and edema of the brain, focal hemorrhagic necrosis of the renal cortex, along with hydropic degeneration of the liver and kidneys. Commonly, calcium oxalate crystals are found within the CNS (brain and spinal cord) and kidneys.[18] The primary pathway of or to exposure, excluding direct contact, is via contact with contaminated water or soil.[12]

2,4,6-Trinitrotoluene

As is commonly known, trinitrotoluene is used as an explosive. The acute fatal dose is between 1-2g; while the exposure limit is $0.5mg/m^3$.[20] In an exposed organism, TNT injures almost every cell it contacts; in particular, those cells of the liver, bone marrow and kidney. Pathological findings of a TNT poisoning would most likely tell of acute, yellow atrophy of the liver, bone marrow aplasia, petechial hemorrhages and toxic nephritis. Bone marrow involvement is communicated via laboratory findings of depressed RBC counts, in conjunction with anisocytosis and poikilocytosis; there may be relative lymphocytosis, as well.[20]

TNT enters the environment via waste-waters and solid waste products of the armament industry. This compound, like so many others, is able to, and all too frequently does, migrate via surface water and soils into groundwater. Trinitrotoluene also displays bioaccumulative capabilities; with the most likely route of exposure being contact with contaminated surface and/or ground waters.[12]

Produce Pesticides/Paternal Exposure

Paternal chemical exposure via three decades of produce handling includes, but may not be exclusive to, the following chemicals. Please note that following each chemical is a list of associated health effects linked to exposure to that particular chemical.

Acephate-found on cranberries

·carcinogenic; damages brain and nervous system

Azinphos Methyl-found on apples

·damages brain and nervous system

Captan-found on strawberries

·carcinogen; damages reproductive system; causes birth defects; damages brain and nervous systems; damages the immune system

Carbaryl-found on peaches and oranges

·carcinogen; damages reproductive system; causes birth defects; damages brain and nervous system; interferes

with hormones

Chlordane-*Cis*-found on summer squash and winter squash

-carcinogen; damages reproductive system; causes birth defects; damages brain and nervous system; interferes with hormones

Chlordane-*Trans*-found on summer squash and winter squash

-carcinogen; damages reproductive system; causes birth defects; damages brain and nervous system; interferes with hormones

Chlorothalonil-found on string beans and onions

-carcinogen; damages brain and nervous system

Chlorpyrifos-found on peaches

-damages brain and nervous system

DCPA-found on broccoli, turnip greens, turnips, lettuce romaine

-carcinogen

DDE-found on spinach and potatoes

-carcinogen; damages reproductive system; causes birth defects; damages brain and nervous system; interferes with hormones

DDE, P,P[1]-found on broccoli, turnip greens, lettuce romaine

-carcinogenic; damages reproductive system; causes birth defects; damages brain and nervous system;

interferes with hormones

DDT-found on spinach

-carcinogen

Dicloran-found on peaches

-health effects unknown

Dieldrin-found on winter squash

-carcinogen; damages reproductive system; damages brain and nervous system; interferes with hormones; damages the immune system

Endosulfan 1-found on summer squash

-damages brain and nervous system; interferes with hormones

Endosulfan 2-found on summer squash

-damages brain and nervous system; interferes with hormones

Endosulfan Sulfate-found on watermelons, cucumbers, summer squash, winter squash

-damages brain and nervous system; interferes with hormones

Ethion-found on grapefruit

-damages the brain and nervous system

Imazalil-found on bananas and oranges

-carcinogen; causes birth defects; damages brain and nervous system

Iprodine-found on peaches

-carcinogen

Methamidophos-found on string beans and tomatoes

-damages brain and nervous system

Omethoate-found on tomatoes

-health effects unknown

Oxamyl-found on tomatoes

-health effects unknown

Permethrins-found on spinach and tomatoes

-carcinogen; interferes with hormones

Thiabendazole-found on potatoes, apples, bananas, grapefruit

-causes birth defects; damages brain and nervous system

Trifluralin-found on carrots

-carcinogen; damages reproductive system; causes birth defects; interferes with hormones; damages immune system[21]

Of these 26 chemicals, many are classified as cholinesterase inhibitor pesticides: acephate, azinphos, chlorpyrifos, ethion, omethoate-are

recognised as being organic phosphates; whereas, carbaryl and oxamyl are recognised as carbamates.[22]

Cholinesterase inhibitors are most commonly employed in agriculture to control soft-bodied insects. The organophosphorous derivatives act via combining with and subsequently inactivating acetylcholinesterase.[23] This combination is believed to occur according to the following reaction:

$$AChe + (RO)_3PO \Rightarrow ROH + (RO)_2PO(AChe) \Rightarrow (RO)_2PO(OH) + Ache$$

The pace of this reaction and stability of product, the cholinesterase-phosphate combination, are rather dependent upon the structure of the phosphate ester.[22]

The action of the carbamates is similar in mechanism, although the combination is reversible.[22]

The inactivation of cholinestrase, by these pesticides, permits acetylcholine to accumulate. This neurotransmitter build-up is not without consequence; it contributes to a rather complex sequelae. First, there exists the possibility of/for the potentiation of postganglionic parasympathetic activity; such CNS activity is corporeally expressed as: constricted pupils, stimulation of intestinal muscles along with salivary and sweat glands; constriction of bronchial muscles, contraction of the urinary bladder, slowing of the sinus node and blockage of the AV node. This initial excitation is followed by the extended depolarization of the skeletal muscles; ultimately resulting in paralysis. In conjunction, there is a

depression of the CNS, precipitating inhibition of the inspiratory center- effectively terminating respiration. The final component is variable ganglionic stimulation or blockage, expressed as either a rise or fall in bp and/or dilation or constriction of the pupils.[23]

In addition to the cholinesterase inhibitor pesticides, the aforementioned 26 pesticides also include a number of chemicals that are recognised as endocrine/hormone disrupters; including carbaryl, DDT, metabolites of DDT, dieldrin, endosulfan, permethrin and trifluralin.[24]

Hormone or endocrine disrupters are chemicals recognised to have the ability to interfere with the endocrine system of animals and humans; the compounds are able to block or even mimic the body's natural hormone signals. Thereby sending false hormone messages, interrupting real hormone messages, preventing the synthesis of the body's true hormones, and even accelerating the degradation and elimination of the true hormones. Obviously then, a number of health effects have been associated with endocrine disrupters, including: reproductive disorders, dysfunction of the immune system, cancer (breast, prostate, testicular), neurological effects, attention deficit and compromised short-term memory, decreased/low IQ's. Furthermore, it has been suggested that these chemicals may pose a very specific threat to both the developing fetus and young children; with exposure in-utero and via breast milk.[24]

It is just such early chemical exposure that is believed to be contributing to some rather disturbing trends in childrens' health, and in the reproductive health of adults. Please consider the following:

1. Childhood cancers, cancers in children <15 years of age, have risen 10% between 1974 and 1991 in the United States; cases of ALL-acute lymphoblastic leukemia, rose by 1% per year in the US from 1973 to 1994. The rate of brain cancer has increased 2% per year during the same time frame.[25]
2. A number of studies have confirmed the trend of American girls entering puberty earlier than was found in past research. There is believed to be a chemical contribution to this change: for in a recent study, it was substantiated that girls, whose mothers had the highest level of PCB's and DDE in their system while pregnant, entered puberty 11 months earlier than girls whose mothers had significantly lower levels of the pesticides.[26]
3. The ratio of male to female births has dramatically declined in recent decades. Although a number of theories of explanation have been offered, parental exposure to endocrine disrupters appears most likely. The endocrine disrupter theory is supported by a study conducted in Seveso, Italy; where large volumes of dioxin were released into the environment, following an industrial accident. Eight years after the

accident, 12 daughters and 0 sons were born to nine couples recognised to have had the highest levels of dioxin exposure.[27]

4. Testicular cancer has increased an astonishing 55% in England and Wales between 1979 and 1991; with the diagnosis of 1,137 new cases in 1991 alone. The development of this particular form of cancer is believed to be strongly influenced by developmental aberrations of the testes in utero-with endocrine disrupters suspected as initiating such aberrations.[28]
5. Oligospermia or decreased sperm count, is becoming increasingly common in men of all age brackets, throughout Europe and the US.[28]
6. Breast cancer has been on the increase 1% per year since the 1940's in the US; and between 1945 and 1980, Denmark experienced a 50% increase in this form of cancer. A number of studies have drawn a direct relationship between breast cancer and exposure to endocrine-disrupting chemicals-such as DDT, dioxin and PCB's.[28]
7. In England and Wales, prostate cancer has increased 40% from 1949 to 1991.[28]

Chapter 6: The Genetics

It has been suggested that every recognized cancer is the result of some genetic event or better yet, genetic damage. Of course, implicit to this statement is the recognition that assaults upon an individual's DNA are possible via x-rays/radiation, chemicals-misperscribed/misadministered pharmaceuticals-environmental pollutants, and viruses; for there must be some sentinel event that initiates this "cascade." When studying the cytogenetics of this particular case of AML-M2, it is not unlikely that genetic aberrations were integral in allowing disease progression (Appendix 1-cytogenetic data).

The anomalies for this leukemia patient include the following:

1. consistent hypodiploidy;
2. random, nonclonal chromosomal loss;
3. loss of Y-sex chromosome;
4. translocation between the long arms of chromosome 11 at 11q13 and 15 at 15q22;
5. translocation involving the long arms of chromosome 8 at 8q24.1 and Y at Yq12;
6. the presence of two cell lines within the bone marrow.

Table 6-1

Glossary of Cytogenetic Terminology[29]

Centromere-The constriction along the length of the chromosome that is the site of the spindle fibre attachment. The position of the centromere dictates whether chromosomes are X-shaped (**metacentric**) or V-shaped (**acrocentric**).

Karyotype-Arrangement of chromosomes from a particular cell according to an established system such that the largest chromosomes are first and the smallest ones are last. A normal female karyotype is represented as 46, XX; a normal male karyotype is represented as 46, XY.

Translocation-A break in a minimum of two chromosomes with an exchange of material.

Deletion-A segment of a chromosome goes missing as a result of a single break (**terminal deletion**) or two breaks with loss of the intervening segment (**interstitial deletion**).

Inversion-Two breaks occur in the same chromosome with a rotation of the interim segment. If both breaks occur on the same side of the centromere, it is known as a **paracentric inversion**; if the breaks are on opposite sides, it is known as a **pericentric inversion**.

The genes that are suspected of contributing to the development of leukemia are commonly divided into five classes:

1. those genes that carry growth-stimulating signals from the cell nucleus;
2. genes that activate transcription or RNA synthesis within the nucleus;
3. genes responsible for the promotion of cell differentiation;
4. genes involved in apoptosis-referring here to the programmed cell death experienced by blood cells upon completion of their functions;
5. "anti-oncogenes" or those genes that suppress tumour development, under normal biochemical/genetic conditions.[30]

Table 6-2

Primary Cytogenetic Subgroups in Acute Myelogenous Leukemia[31]

Translocation	FAB/Incidence
t(8;21)	-20% of M2; 6-10% of de novo AML

Clinical Morphology: auer rods, hypergranulated myelocytes, durable remissions.

t(15;17)	-99% of M3; approx 10% de novo AML

Clinical Morphology: consumptive coagulopathy (DIC), durable remissions with all-trans-retinoic acid and additional chemotherapy; hypergranular variant w/coarse azurophilic granulation; microgranular variant with decreased granulation and nuclear constrictions.

Inv(16); t(16;16)	->90% of M4Eo; 7-10% de novo AML

Clinical Morphology: marrow eosinophilia w/coarse irregular basophilic granules.

t(11q23; variable)	-approx 5% de novo M4 and M5 AML approx 5% t-AML

Clinical Morphology: variable morphology but w/a monocytic component; associated w/t-AML and a generally poor prognosis.

Table 6-3

Prognostic Impact of Selected Chromosome Abnormalities in de novo AML

Karyotypic Abnormality	Complete Remission Rate	Length of CR
Inv(3)	low	short
-5/5q	low	short
-7/7q	low	short
t(8;16)	low	short
t(8;21)	high	long
+8	variable	variable
t/del (11q23)	variable	short
t(15;17)	high	long
inv(16)	high	long
+21	high	variable

After digesting the aforementioned genetic information and data, some troubling aspects to this particular case of AML M2 come to the fore.

1. The identification of at least two cell lines indicates that karyotypic evolution has occurred-if the karyotypes are related; however, if the two karyotypes are unrelated, this could indicate

the occurrence of two independent leukemogenic events (Alimena).

Multiple clones occur more frequently in those patients with secondary leukemia (77.9%), compared to patients with ANLL de novo (10.8%). Slightly more than 33% of all cases with multiple clones had losses of part or even all of chromosome #5 and /or chromosome #7-as a first step change. However, 9:10 patients with secondary leukemia and multiple clones had involvement of the chromosomes. The second step chromosomes most often involved include #9, #17 or #21. Those patients found to express t(8;21) in addition to multiple clones, most often had loss of a sex chromosome.[32]

2. There is a subset of ANLL patients, characterized by the presence of t(8;21) in bone marrow cells. At the Second International Workshop on Chromosomes in Leukemia, 40 such patients were reviewed, and it was recognized that:
    A. the occurrence of the translocation was intimately related to the morphologic diagnosis of FABM2 (acute myeloblastic leukemia with maturation);
    B. the loss of a sex chromosome was frequently associated with this translocation;

C. the rate of both remission and survival were recognized as relatively good, most notably in cases with some normal metaphases; whereas the association of a missing sex chromosome with t(8;21) carried a poor prognosis;

D. there was some geographic difference in the occurrence of t(8;21).[33]

Of particular interest in this case: consistent hypodiploidy, loss of the Y-sex chromosome, secondary chromosomal rearrangements consistent with having received chemotherapy, and a relatively low number of observed metaphases. Furthermore, the initial or diagnostic cytogenetics, do not communicate any necessarily inherited (maternal/paternal) chromosomal anomalies (breakpoints, etc); thereby raising the question of what exactly was the sentinel event in this child's life-that could have provoked a leukemogenic event? The possible suspects, in this case, are quite unfortunately becoming the usual suspects in the development of childhood cancers: pharmaceuticals (in-utero, neonate exposure, or both), and the external environment (exposure to contaminated food, water, soil).

Chapter 7: Materials and methods

The portion of this case study concerned with assessing any external environmental influence was initiated on 10.22.98, 12 days after the patient's admission to hospital; under the original diagnosis of pancytopenia/aplastic anemia. This data was collected over seven days; and includes water samples from Berlin Lake (Appendix 3), and soil samples from the lawn of the patients home (Appendix 3).

Materials

All of the solvents used within the context of this study were pesticide grade (Fisher Scientific, Fairlawn, NJ). Additional reagents included: 100-mesh silicic acid (Mallinckrodt Chemical Works, St. Louis, MO); 80-200 mesh alumina, anhydrous sodium sulfate (Fisher Scientific, Fairlawn, NJ) and sodium chloride (VWR Scientific, Westchester, PA). The analytical standards used within this case-study, were purchased from Supelco, Inc (Bellefonts, PA) or Ultra Scientific (North Kingstown, RI). The helium and nitrogen were ultra pure carrier grade. The filters employed for the preparation of the water samples were type GMF grade filters, 47mm, (Whatman, Maidstone, England); the accompanying polyurethane foam plugs were purchased from Graseby Anderson (Cleveland, OH).

The water filters were precleaned via baking at 450°C for a minimum of 20h in a muffle furnace; they were then wrapped in 'cleaned' aluminum foil and sealed in plastic bags. The polyurethane foam plugs were prepared for use via soxhlet extraction, 18h in acetone; followed with soxhlet extraction in petroleum ether, 18h. Upon completion of the cleaning protocol, the polyurethane foam plugs were dried, via low heat, in a dry seal dessicator; following which they were stored in glass jars with teflon-lined lids. The silicic acid was 'cleaned' via baking at 140°C, 24h; and before its use, it was deactivated with 1.7% water. The adsorption alumina was 'cleaned' via overnight baking at 450°C; prior to its use, it was deactivated with 6% water and stored in a glass jar with a teflon-lined lid. The anhydrous sodium sulfate was prepared in a similar manner, with overnight baking at 450°C and stored in a glass jar with a teflon-lined lid. Any sodium chloride crystals used, were prepared via a petroleum ether rinse, followed-up with a dichloromethane rinse; and then dried at 140°C. Boiling chips, used throughout this protocol, were prepared by soxhlet extraction with petroleum ether in a cellulose thimble, 12h; they were then dried at 140°C and stored in a glass jar with a teflon-lined lid.

Sample Collection and Work-up

Soil

The lawn sample was collected 10.22.98; stored in cleaned aluminum foil within a labeled plastic bag at 4°C until further work-up was possible.

The soil, was later thawed and manually mixed, to promote homogeneity. Approximately 15.721g of the soil was mixed with sodium sulfate to remove any water. The dried soil was spiked with 452ng PCB-103, transferred to a 'clean' cellulose thimble and extracted, via soxhlet with dichloromethane, 24h. The extract was then reduced via rotary evaporation, transferred into hexanes and concentrated under nitrogen, to 2ml.

The extract was then cleaned via an alumina column composed of a glass wool plug, on top of which was 2g Al_2O_3 and 1cm Na_2SO_4. The alumina column was pre-prepared with 5ml of 5% dichloromethane in petroleum ether. The sample was then added to the column and eluted with 20ml 5% dichloromethane in petroleum ether. The resulting eluent was then concentrated and solvent exchanged into iso-octane under nitrogen.

Water

Water samples were collected in cleaned 4L solvent jugs, from locations in and around Berlin Lake (Appendix 3). The samples were stored at 4°C until extraction was possible.

The polyurethane foam plugs were extracted via soxhlet, in petroleum ether, for 24H. The filters, prior to use, were refluxed in dichloromethane for 18h.

The water samples were transferred into individual stainless steel canisters (Coca-Cola Bottling Company of Northern Ohio, Youngstown, Ohio). The water samples were pushed, via nitrogen pressure, through a 47mm GMF water filter, in attempt to remove any particulate matter; each sample required several filters, due to high levels of particulate matter. The water filters were then wrapped individually in cleaned aluminum foil and stored in plastic bags at -10°C.

The filters were soxhlet extracted with dichloromethane 24h. The extracts were then reduced to 5-10ml and solvent exchanged into iso-octane via rotary evaporation. The entire sample inventory was reduced individually to 1ml under nitrogen. The samples were cleaned using a silicic acid/alumina column; a glass column was dry-packed with a first layer of 3g silicic acid (1.7% water added), followed by a second layer of 2g adsorption alumina (6% water added), and a third layer of 2cm anhydrous sodium sulfate.

Chapter 8: Results

The spectra, as collected for this particular case study, may be found in Appendix 4. The following analyses of the data were made possible through these references: (it is important to note that IR analysis done in this manner is non-specific)

- The IR Wizard on the web;[34]
- *Spectrometric Identification of Compounds 5th ed.* Appendix C: Characteristic Group Absorptions. R.M. Silverstein, G.C. Bassler, J.C. Morrill; John Wiley and Sons, Inc. New York:1991.[35]

K-Soil

*collected 10.22.98

*sample run 4.29.99

*peaks of interest: 1300-1050 cm^{-1} possible functional groups include esters and/or lactones;

2900 cm^{-1} possible functional groups include –CHO, -CH_3, -CH_2.

K-soil

*collected 10.22.98

*sample run 4.30.99

*peaks of interest: 1300-1050 cm^{-1} possible functional groups include esters and/or lactones;

2900 cm^{-1} possible functional groups include $-CH_3$, $-CH_2$.

Filter 3-B Feed

*collected 10.22.98

*sample run 5.3.99

*peaks of interest: 2354 cm^{-1} possible functional groups include $-NH_2^+$, $-NH^+$, $=NH^+$, P-H.

Syringe B Feed

*collected 10.22.98

*sample run 5.3.99

*peaks of interest: 2945.6 cm^{-1} possible functional groups include $-CH_3$, $-CH_2$;

1100 cm^{-1} possible functional groups include P-O-alkyl, -COH, ROCOCOR, C=S, S=O.

Filter 4 B Feed

*collected 10.22.98

*sample run 5.2.99

*peaks of interest: 2400 cm^{-1} possible functional groups include NH_2^+, NH^+, $=NH^+$, P-H.

Filter 2 B Feed

*collected 10.22.98

*sample run 5.3.99

*peaks of interest: 2350 cm^{-1} possible functional groups $-NH_2^+$, NH^+, P-H.

Filter 1 B Feed

*collected 10.22.98

*sample run 5.2.99

*peaks of interest: 2900 cm^{-1} possible functional groups include $-CH_3$, $-CH_2$;

500 cm^{-1} possible functional groups include C-I alkyl.

C-Feed

*collected 10.25.98

*sample run 4.30.99

*peaks of interest: 2900 cm^{-1} possible functional groups include –CHO, $-CH_3$, $-CH_2$;

1300-1050 cm^{-1} possible functional groups include esters and/ or lactones.

Filter 1 B Feed

*collected 10.25.98

*sample run 5.4.99

*peaks of interest: 2250 cm^{-1} possible functional groups include aromatic ketones;

1640 cm^{-1} possible functional groups include o-amino or o-hydroxyarylketones, 6-membered rings (-NCON-).

Filter 2 B Feed

*collected 10.25.98

*sample run 5.3.99

*peaks of interest: 2960 cm^{-1} possible functional groups include $-CH_3$, $-CH_2$

Section A/Dam Water

*collected 10.25.98

*sample run 5.18.99

*peaks of interest: 2250 cm^{-1} possible functional groups include aromatic ketones;

1640.9 cm^{-1} possible functional groups include o-amino or o-hydroxyarylketones, 6-membered rings (-NCON-).

Dam Water

*collected 10.25.98

*sample run 5.18.99

*peaks of interest: 2250 cm^{-1} possible functional groups include aromatic ketones;

1640.9 cm^{-1} possible functional groups include o-amino or o-hydroxyarylketones, 6-membered (-NCON-).

Beyond Dam

*collected 10.25.98

*sample run 5.4.99

*peaks of interest: 2080.9 cm^{-1} possible functional groups include –N=C=S;

1640.9 cm^{-1} additional possibilities include C=N

(conjugated cyclic), C=N and/or –C=C-C=N-.

C Feed

*collected 10.25.98

*sample run 4.29.99

*peaks of interest: 2950 cm^{-1} possible functional groups include $-CH_3$, -

CH_2;

1500-1030 cm^{-1} possible functional groups include

esters and/or lactones.

Syringe C Feed

*collected 10.25.98

*sample run 5.3.99

*peaks of interest: 2950 cm^{-1} possible functional groups include $-CH_3$, -

CH_2;

1050 cm^{-1} possible functional groups include P-O-alkyl,

C=S, S=O, C-OH, ROCOCOR.

Please note when reading the data collected, that the main objective of the collection/analysation of such samples was to provide further evidence for the presence of these chemicals; these chemicals/functional groups are already recognised contaminants of the Mahoning River Basin (EPA). Therefore, this summary is presented to merely lend support to the casual correlation between chemical contaminants and AML. It is this correlation that is being discussed within this thesis.

Chapter 9: Concluding Remarks

It would be virtually impossible, as well as completely academically irresponsible, to compose a definitive statement as to how this case of pediatric AML-M2 arose; however, as this thesis is a single case-study, it is able to contribute several documented casual-although perhaps not so casual-links to chemical exposure and increased risk for leukemogenesis

If the reader goes beyond the necessarily limited focus of this single case-study, he/she will be able to find scores of articles/recent publications that are able to claim direct links between chemicals and cancer. Thus, if this study's documentation should be met with any degree of incredulity, it may behoove the reader to consider the following:

1. Children whose homes and/or yards were treated with pesticides are believed to be at a greater risk for developing childhood cancer, according to a study published February 27, 1995 in The American Journal of Public Health. The researchers, involved with this study, compared home pesticide use in >52 cases of childhood cancer in Denver, CO, between 1976 and 1985 with those of 222 healthy children with similar demographic profiles. Children from birth through 14 years of age, whose yards were routinely treated with herbicides and/or insecticdes, had a 4-fold increased risk of soft tissue sarcomas and malignant tumours of the connective tissues.

compared to their healthy contemporaries. Furthermore, the study found that in-utero exposure to pest-strips, during the 3rd trimester of pregnancy, increased a neonate's risk for developing leukemia three times (University of N. Carolina www.enn.com).

2. A review of 61 studies, published in BioEssays 1999, concluded that the sharp decline in average sperm density, in the western world, may be even more dramatic than previously believed. For the University of Copenhagen, 1992, found a 50% decline in sperm density between 1938 and 1990. A later reanalysis of this, conducted by the University of Missouri-Columbia, proposes that the decline most likely exceeds 50%. It is believed that this startling trend may be attributable, in part, to inadequate amounts of available estrogen; realising of course, that estrogen is necessary for the production of healthy sperm (www.cnn.com).
3. High blood levels/concentrations of organochlorines (DDT, DDE, PCB) have been associated with gene mutations identified in patients diagnosed with pancreatic cancer. Patients with a formal diagnosis of pancreatic cancer were 5-10 times more likely to display elevated organochlorine blood levels-compared to those patients hospitalized for medical conditions other than cancer. Additionally, concentrations of both DDT and DDE were most elevated in those patients expressing mutations in the gene K-ras; K-ras is suspected

of being a target for carcinogens. In Spain, where this study was conducted, 78-100% of analysed meat samples were found to contain DDE; while 50% of the fish samples contained PCB (Lancet 1999; 354:2125-2129).

4. The organophosphate pesticide, chlorpyrifos, is one of 40 such compounds, currently being reviewed by the EPA; in attempt to determine the health risks it may represent, primarily for children. Chlorpyrifos, produced by Dow Chemical Co., is recognised on the market as Dursban and/or Lorsban; Dursban and Lorsban are found in over 800 products with applications inside of homes and hospitals. The EPA estimates that 20 million to 24 million pounds of this chemical are applied annually. A recent study of 993 adults found that 8 in 10 urine samples contained quantifiable amounts of chlorpyrifos. Even more alarming, is the finding that of 89 children studied, 9 of 10 urine samples tested positive for chlorpyrifos-in quantifiable amounts, as well (www.MSNBC.com).

The ubiquitous character of chemical contamination is absolutely alarming; misplaced, misused chemical compounds dominate our lives and more importantly, the lives of our children. Forty percent of all human deaths are directly linked to some sort of environmental influence: radiation, air pollution, soil pollution, organochlorines, endocrine disrupters. To deny this control that chemical contaminants exact upon

the globe is simply foolhardy. Quite simply then, exposure to chemicals damages DNA; and damaged DNA misreads coding signals; genetic misreads precede genetic mutations and genetic mutations precede cancer-this much is indisputable.

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APPENDIX 1

Medical Data: Cytogenetics, Flow cytometry reports, Surgical pathology reports, Hematopathology reports

CYTOGENETICS

Forum HEALTH

NORTHSIDE MEDICAL CENTER
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BEEGHLY MEDICAL PARK

DEPARTMENT OF LABORATORY MEDICINE

CYTOGENETICS AND MOLECULAR GENETICS LABORATORY
(330) 740-3765 / 3756

CYTOGENETICS REPORT

PATIENT [redacted]
DATE OF BIRTH :5/31/89
HOSPITAL NUMBER :0272192
ACCESSION NUMBER :10-11-231M-98
LOCATION :[redacted]
DOCTOR :[redacted]
REFERRAL :Pancytopenia/ AML

SPECIMEN TYPE :Bone Marrow
SPECIMEN COLLECTION DATE : 10/11/98
SPECIMEN RECEIVED DATE :10/11/98
PRELIMINARY DATE :11/9/98
FINAL DATE :11/10/98

STAINING METHOD :GTG
CULTURES ANALYZED :4
CELLS KARYOTYPED :4
RESOLUTION :475 Bands

<45	45	46	47	>47	Total
5	4	11	0	0	20

CYTOGENETIC DIAGNOSIS :45,X,-Y[3]/46,XY[17]

COMMENTS:

All observations were made from direct, overnight, and T-cell and B-cell stimulated cultures. Two cell lines were detected in this specimen. The first cell line (3/20) contained a modal number of 45 chromosomes including one X chromosome. However, each cell in this line was missing the Y chromosome. Although loss of the Y has been shown to be a normal age-related phenomenon in older males, this finding is not common in a patient of this age. Loss of the Y has been described in AML, often as a secondary change. The second cell line 17/20) was the normal male karyotype.

[redacted], Scientific Director, Cytogenetics

[redacted], Medical Director, Cytogenetics

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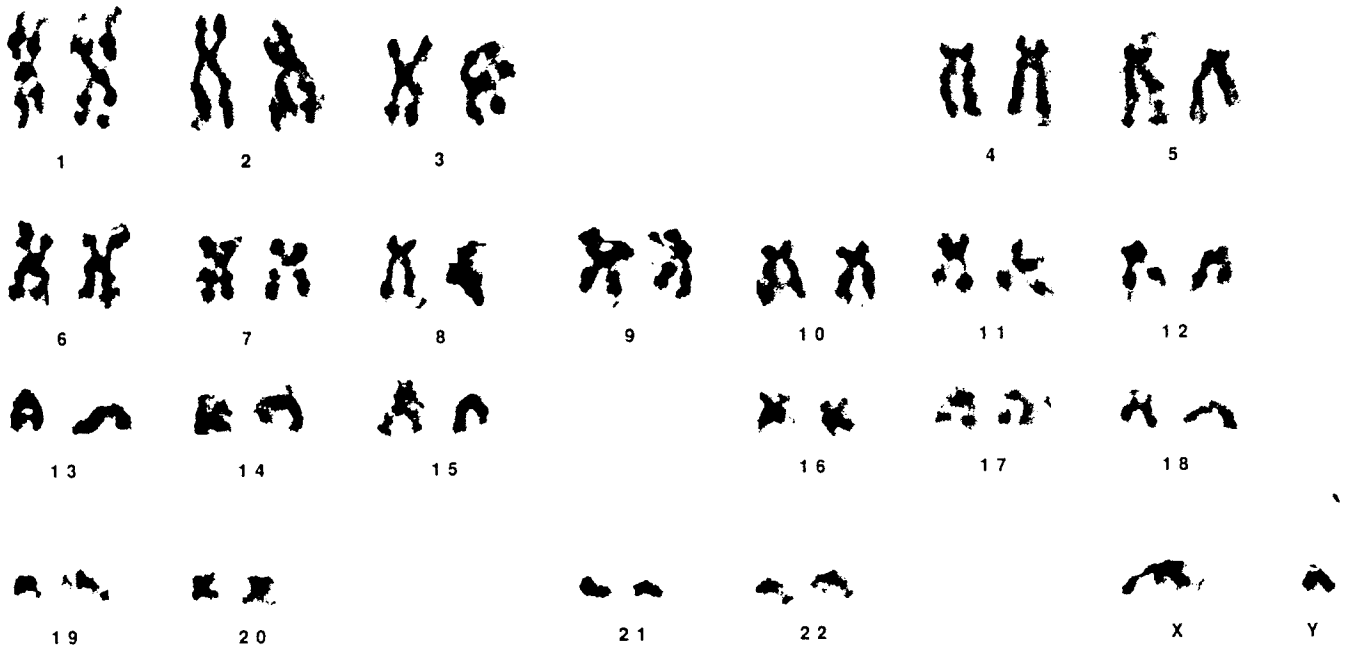
Department of Cytogenetics

Patient Name:
Accession No.:10-11-231M-98
Karyotype Designation:
Date of Birth:5/31/89
Referring Doctor: [redacted]
Doctor Drawn: [redacted]
Slide List:6A 173.7x4.6 (2) kary
Resolution:500 Bands

Forum Health / Northside Medical Center
Department of Cytogenetics

atient Name:I _ecimen #3)
:cession No.:11-9-252M-98
aryotype Designation:46,XY[1]
ate of Birth:5/31/89
eferring Doctor:[redacted]
octor Drawn:
.ide List:8A 137.5x22.1(1)kary
esolution:500 bands



CHILDREN'S CANCER GROUP
CYTOGENETICS REPORTING FORM

To be completed by the Institutional Cytogeneticist and submitted to the Group Operations Center.

PLACE LABEL HERE
STUDY ID: 2961 - E - 10
REG #: 50095
PT NAME: K[redacted]

(E/10)

[ ] Sex
1= Male
2= Female

Lab case No.: _____

Date/time specimen collected: [1][1][0][9][9][8] (M M D D Y Y) 1230 AM/PM

Date/time specimen received: [1][1][0][9][9][8] (M M D D Y Y) 130 AM/PM

Type of specimen: check all that apply (fill out separate form for each type of tissue)

- [✓] bone marrow aspirate
- [ ] bone marrow biopsy
- [ ] peripheral blood
- [ ] lymph node
- [ ] other (specify) _____

If unsatisfactory results, check boxes

- [✓] poor sample (clotted, hypocellular, contaminated, etc.) 1.5 cc; clotted
- [✓] interphase nuclei present but few or no metaphases
- [ ] poor quality metaphases and/or inadequate banding (Note: even though unsatisfactory results, please fill in the processing information on the back of the page).

Note: Even if the study was inadequate, please fill in the processing information on the back of this page.

Additional Comments

Only 1 metaphase observed in 10 cultures.
Patients first tap was clonal 45,X-Y /46,XY ; 2nd tap contained fewer metaphases (46,XY [13]).

Forum Health - Trumbull Memorial Hospital
Name of Institution

[redacted] 11/2/01
Cytogeneticist/Date

CHILDREN'S CANCER GROUP
CYTOGENETICS REPORTING FORM

PLACE LABEL HERE
STUDY ID: 2961 - E - 10
REG #: 60095
PT NAME.
(E/10)

PROCESSING INFORMATION (Fill in the number of cells obtained from each processing method A, B, C, or D.)

	A	B	C	D	TOTAL NO.
NORMAL	0	0	1		1
CLONE-1					
CLONE-2					
CLONE-3					
ABNORMAL NONCLONAL					
TOTAL NO.	0	0	1	0	1

Specify type of processing and the type of banding used for each of the lettered boxes above.

A. Direct - GTG
B. Overnigh - GTG
C. B-cell stimulated - GTG
D. T-cell stimulated - GTG

List karyotypes of each clonal and nonclonal cell (ISCN 1995. DO NOT include cells with random loss as nonclonal abnormal cells.)

NORMAL: 46, XY [1]
CLONE-1:
CLONE-2:
CLONE-3:
NONCLONAL-1:
NONCLONAL-2:
NONCLONAL-3:

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CYTOGENETICS REPORT

PATIENT
DATE OF BIRTH : 5/31/89
HOSPITAL NUMBER : 0272192
ACCESSION NUMBER : 11-23-268M-98
LOCATION : [redacted]
DOCTOR : [redacted]
REFERRAL : AML

SPECIMEN TYPE : Bone Marrow
SPECIMEN COLLECTION DATE : 11/23/98
SPECIMEN RECEIVED DATE : 11/23/98
PRELIMINARY DATE : 12/9/98
FINAL DATE : 12/10/98

STAINING METHOD : GTG
CULTURES ANALYZED : 1
CELLS KARYOTYPED : 1
RESOLUTION : 525 bands

<45	45	46	47	>47	Total
0	0	1	0	0	1

CYTOGENETIC DIAGNOSIS : 46,XY[1]

COMMENTS:

Ten cultures were initiated on this specimen including direct, overnight and T- and B-cell stimulated cultures. Only one metaphase was observed in the T-cell stimulated culture and was apparently the normal male karyotype. However, due to poor growth of the specimen, the possibility of chromosomal mosaicism involving abnormal cell lines cannot be excluded.

[redacted], Scientific Director, Cytogenetics

[redacted], Medical Director, Cytogenetics

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CYTOGENETICS REPORT

'ATIENT
)ATE OF BIRTH :5/31/89
IOSPITAL NUMBER :0272192
\CCESSION NUMBER : 12-9-277M-98
.OCATION [redacted]
)OCTOR : [redacted]
\EFERRAL : AML

SPECIMEN TYPE : Bone Marrow
SPECIMEN COLLECTION DATE : 12/9/98
SPECIMEN RECEIVED DATE : 12/9/98
PRELIMINARY DATE : 1/7/99
FINAL DATE : 1/11/99

STAINING METHOD : GTG
CULTURES ANALYZED : 3
CELLS KARYOTYPED : 4
RESOLUTION : 550 bands

Cells counted

<45	45	46	47	>47	Total
0	6	16	0	0	22

CYTOGENETIC DIAGNOSIS : 46,XY

COMMENTS:

Normal Male Karyotype.
All observations were made from T and B cell stimulated cultures. Please note that although 6/22 cells were hypodiploid, all displayed random, nonclonal chromomal loss.

No chromosome abnormalities were demonstrable at this level of resolution.
Please remember that this analysis does not eliminate the possibility of single cell defects, chromosomal mosaicism involving abnormal cell lines of low frequency or small chromosomal structural abnormalities.

[redacted], Scientific Director, Cytogenetics

[redacted], Medical Director, Cytogenetics

Forum Health / Northside Medical Center
Department of Cytogenetics

Patient Name [redacted]
Accession No.:12-9-277M-98
Karyotype Designation:46,XY
Date of Birth:5/31/89
Referring Doctor:[redacted]
Doctor Drawn:
Slide List:6A 152.4x10.5kary(2)
Resolution:550 bands

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CYTOGENETICS REPORT

PATIENT : [redacted]
DATE OF BIRTH :5/31/89
HOSPITAL NUMBER :0272192
ACCESSION NUMBER : 12-30-294M-98
LOCATION [redacted]
DOCTOR : [redacted]
REFERRAL : AML

SPECIMEN TYPE : Bone Marrow
SPECIMEN COLLECTION DATE : 12/30/98
SPECIMEN RECEIVED DATE : 12/30/98
PRELIMINARY DATE :1/15/99
FINAL DATE :1/18/99

STAINING METHOD : GTG
CULTURES ANALYZED : 2
CELLS KARYOTYPED : 4
RESOLUTION : 550 Bands

Cells counted

<45	45	46	47	>47	Total
0	3	17	0	0	20

CYTOGENETIC DIAGNOSIS : 46,XY

COMMENTS:

Normal Male Karyotype. All observations were made from T cell stimulated cultures.

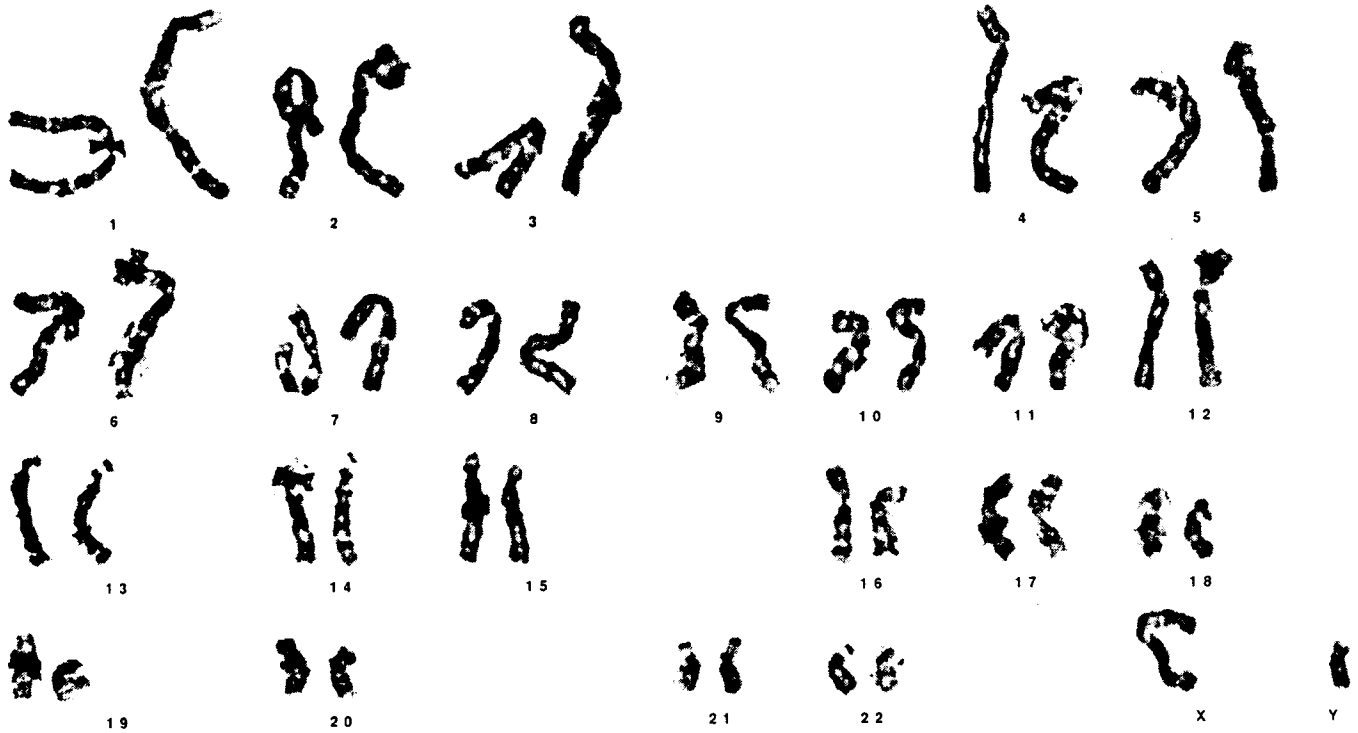
No chromosome abnormalities were demonstrable at this level of resolution.
Please remember that this analysis does not eliminate the possibility of single cell defects, chromosomal mosaicism involving abnormal cell lines of low frequency or small chromosomal structural abnormalities.

[redacted] Scientific Director, Cytogenetics

[redacted], Medical Director, Cytogenetics

Forum Health / Northside Medical Center
Department of Cytogenetics

Patient Name: [redacted]
Accession No.:12-30-294M-98
Karyotype Designation:46,XY
Date of Birth:5/31/89
Referring Doctor: [redacted]
Doctor Drawn:
Slide List:6A 129.2x5.8 (2) kary
Resolution:575 Bands



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CYTOGENETICS REPORT

PATIENT [redacted]
DATE OF BIRTH : 5/31/89
HOSPITAL NUMBER : 0272192
ACCESSION NUMBER : 2-18-37M-99
LOCATION : Pediatric Oncology
DOCTOR : [redacted]
REFERRAL : AML

SPECIMEN TYPE : Bone Marrow
SPECIMEN COLLECTION DATE : 2/18/99
SPECIMEN RECEIVED DATE : 2/18/99
PRELIMINARY DATE : 3/18/99
FINAL DATE : 3/18/99

STAINING METHOD : GTG
CULTURES ANALYZED : 8
CELLS KARYOTYPED : 6
RESOLUTION : 550 Bands

Cells counted

<45	45	46	47	>47	Total
2	1	17	0	0	20

CYTOGENETIC DIAGNOSIS : 46,XY

COMMENTS:

Normal Male Karyotype.
All observations were made from direct, overnight and T and B cell stimulated cultures. Although not clonal, several structural abnormalities were detected in the T-cell stimulated cultures which included: one cell with an apparent translocation between the long arms of chromosomes 11 (at band 11q13) and 15 (at band 15q22) and loss of the Y chromosome; and one cell with a translocation involving the long arms of chromosomes 8 (at band 8q24.1) and Y (at band Yq12). Rearrangements involving chromosomes 8 and Y are often seen as secondary changes in AML, and are not specific to any particular FAB subtype. Clinical correlation is necessary.

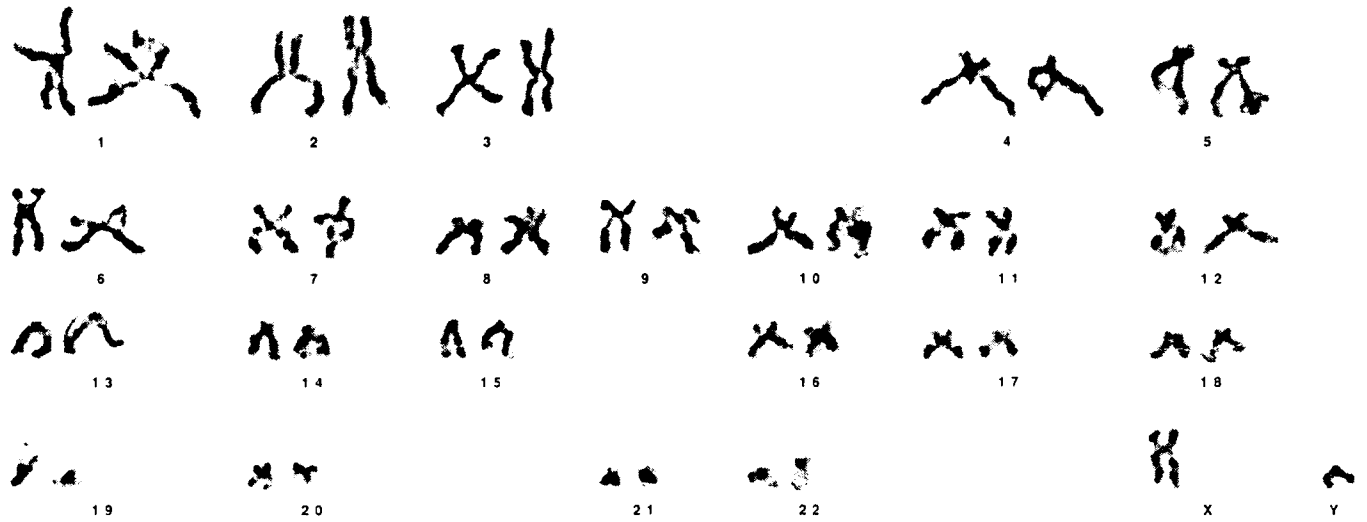
No chromosome abnormalities were demonstrable at this level of resolution.
Please remember that this analysis does not eliminate the possibility of single cell defects, chromosomal mosaicism involving abnormal cell lines of low frequency or small chromosomal structural abnormalities.

[redacted], Scientific Director, Cytogenetics

[redacted] Medical Director, Cytogenetics

Forum Health / Northside Medical Center
Department of Cytogenetics

Patient Name.
Accession No.:2-18-37M-99
Karyotype Designation:46,XY
Date of Birth:5/31/89
Referring Doctor:[redacted]
Doctor Drawn:
Slide List:1A 156.3x20.6(2).kary
Resolution:550 Bands



FLOW CYTOMETRY REPORTS

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DEPARTMENT OF LABORATORY MEDICINE

SAMPLE COPY

FLOW CYTOMETRY CONSULTATION

PATIENT: [redacted]
ADDRESS: [redacted]
[redacted]
AGE/SEX: 9Y M
BIRTHDATE: 05/31/1989
SOC SEC: [redacted]

ACCESS NO: F98-0460
REFERENCE NO: S98-13175
HOSP NO: 0272192
ACCOUNT NO: 002721920011

PAT LOC: T307B (TOD 3)
CPT CODE: 88180 X7

SURGEON: [redacted]
RESIDENT:
ATTEND PHY: [redacted]
COPY TO PHY:

DATE OF OPERATION: 10/11/98
DATE OF ACCESSION: 10/11/98
DATE OF REPORT: 10/17/98

--JH

PREV ACC: F98-0476 S98-13470 S98-13175 X98-3088 S94-07494 S89-09860

SPECIMEN SUBMITTED: A: BONE MARROW

Number of cells examined: 0.12 million/cc

B-cell Antigens	% positive
CD19	22
Kappa light chain	Non-specific binding
Lambda light chain	Non-specific binding

T-cell Antigens	% positive
CD2	56
CD3	35
CD5	38
CD7	70

Myeloid Antigens	% positive
CD13	16
CD14	11
CD33	39

Other Antigens	% positive
CD45	100
CD10 (CALLA)	1
CD34	23

Dual positivity	% positive
CD19 and CD10	1
CD34 and CD7	22

Description: Marrow smears show small numbers of hematopoietic cells with dyspoietic features. Light scatter studies show indistinct marrow cell population with decreased maturation of myeloid cells. A distinct and moderately heterogeneous lymphoid population is seen.

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FLOW CYTOMETRY CONSULTATION

PATIENT: [redacted]
ADDRESS: [redacted]
AGE/SEX: 9Y M
BIRTHDATE: 05/31/1989
SOC SEC: [redacted]

ACCESS NO: F98-0476
REFERENCE NO: S98-13470
HOSP NO: 0272192
ACCOUNT NO: 002721920011
PAT LOC: T307B (TOD 3)
CPT CODE: 88180 X5

SURGEON: [redacted]
RESIDENT:
ATTEND PHY: [redacted]
COPY TO PHY:

DATE OF OPERATION: 10/16/98
DATE OF ACCESSION: 10/16/98
DATE OF REPORT: 10/16/98
--jt

PREV ACC: S98-13470 S98-13175 X98-3088 S94-07494 S89-09860

SPECIMEN SUBMITTED: A: LEFT RETROPERITONEAL LYMPH NODE, NEEDLE BIOPSY

Viability: 90%
Method:

Number of cells examined: 0.05 million/cc

B-cell Antigens	% positive
CD19	0.4
Kappa light chain	0.2
Lambda light chain	0.1

Other Antigens	% positive
CD45	8
CD10 (CALLA)	0.6

Dual positivity	% positive
CD19 and CD10	0.3

Description: Cell suspension shows few lymphoid cells intermixed with moderate amount of red blood cells. Light scatter studies show no distinct lymphoid population.

INTERPRETATION:
LEFT RETROPERITONEAL LYMPH NODE, NEEDLE BIOPSY, FLOW CYTOMETRIC IMMUNOPHENOTYPIC ANALYSIS, MONOTYPIC LYMPHOID POPULATION NOT IDENTIFIED, SEE COMMENT.

COMMENT: Due to small sample size a limited study was performed. No monoclonal lymphoid population was identified. A more adequate sample is recommended for diagnostic evaluation. See also surgical pathology report S98-13470.

[redacted]

jt

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DEPARTMENT OF LABORATORY MEDICINE

FLOW CYTOMETRY CONSULTATION

PATIENT: [redacted]
ADDRESS: [redacted]
AGE/SEX: 9Y M
BIRTHDATE: 05/31/1989
SOC SEC: [redacted]

ACCESS NO: F98-0482
REFERENCE NO:
HOSP NO: 0272192
ACCOUNT NO: 002721920011

PAT LOC: T307B (TOD 3)
CPT CODE: 88180X9

SURGEON: [redacted]
RESIDENT:
ATTEND PHY: [redacted]
COPY TO PHY:

DATE OF OPERATION: 10/20/98
DATE OF ACCESSION: 10/20/98
DATE OF REPORT: 10/22/98

--cw

PREV ACC: S98-13639 F98-0476 S98-13470 F98-0460 S98-13175 X98-3088
S94-07494 S89-09860

SPECIMEN SUBMITTED: A: BONE MARROW AND PERIPHERAL BLOOD

Method: FACS Lyse

Number of cells examined:

B-cell Antigens	% positive	% positive
CD19	3.2	11.6
Kappa light chain	Non-specific	Non-specific
Lambda light chain	Non-specific	Non-specific
T-cell Antigens	**% positive**	**% positive**
CD2	68.1	63.6
CD3	56.5	56.5
CD5	56.4	56.4
CD7	91.5	80.4
Myeloid Antigens	**% positive**	**% positive**
CD13	12.6	14.7
CD14	2.2	9.1
CD33	27.7 (gated)	29.5
Other Antigens	**% positive**	**% positive**
CD45	93.2	99.5
CD10 (CALLA)	1.5	<1
CD34	16.3	14.1

Forum HEALTH
NORTHSIDE MEDICAL CENTER
TOD CHILDREN'S HOSPITAL
BEEGHLY MEDICAL PARK
DEPARTMENT OF LABORATORY MEDICINE

FLOW CYTOMETRY CONSULTATION

PATIENT: I [redacted]
ADDRESS: [redacted]
AGE/SEX: 9Y M
BIRTHDATE: 05/31/1989
SOC SEC: [redacted]

ACCESS NO: L99-0068
REFERENCE NO:
HOSP NO: 0272192
ACCOUNT NO: 002721920094
PAT LOC: PED. HEMONC
CPT CODE: 88180X9

SURGEON: [redacted]
RESIDENT:
ATTEND PHY: [redacted]
COPY TO PHY:

DATE OF OPERATION: 02/18/99
DATE OF ACCESSION: 02/18/99
DATE OF REPORT: 02/19/99
--CW

PREV ACC: F98-0591 S98-17346 X98-3909 F98-0564 S98-16359 F98-0543
S98-15570 X98-3529 X98-3394 R98-00245 F98-0482 S98-13639 F98-0476
S98-13470 F98-0460 S98-13175 X98-3088 S94-07494 S89-09860

SPECIMEN SUBMITTED: A: BONE MARROW

Method: FACS lyse

Number of cells examined:

B-cell Antigens	% positive
CD19	1
Kappa light chain	Non-specific staining
Lambda light chain	Non-specific staining

T-cell Antigens	% positive
CD2	4
CD3	7
CD5	4

Myeloid Antigens	% positive
CD13	6
CD14	8
CD33	18

Other Antigens	% positive
CD45	41
CD10 (CALLA)	<1
CD34	2

Dual positivity	% positive
CD19 and CD10	<1

SURGICAL PATHOLOGY REPORTS

Forum HEALTH
NORTHSIDE MEDICAL CENTER
TOD CHILDREN'S HOSPITAL
BEEGHLY MEDICAL PARK
DEPARTMENT OF LABORATORY MEDICINE

SURGICAL PATHOLOGY REPORT

PATIENT:
ADDRESS: [redacted]
[redacted]
AGE/SEX: 9Y M
BIRTHDATE: 05/31/1989
SOC SEC: [redacted]

ACCESS NO: S98-13175
HOSP NO: 0272192
ACCOUNT NO: 002721920011
PAT LOC: T307B (TOD 3)
CPT CODE: 88305/88311/88313 X2

SURGEON: [redacted]
RESIDENT:
ATTEND PHY: [redacted]
COPY TO PHY:

DATE OF OPERATION: 10/11/98
DATE OF ACCESSION: 10/12/98
DATE OF REPORT: 10/17/98

PRE OP DX: R/O METS.
OPERATION: BIOPSY
POST OP DX: ALL, R/O METS.
HISTORY: POSS. ALL

--ADB

PREV ACC: X98-3088 S94-07494 S89-09860

TISSUES REMOVED: A: LT. PSIS BM BIOPSY AND ASPIRATION

INTRA/EXTRA CONSULT: INTRA

FINAL DIAGNOSIS:

BONE MARROW, BIOPSY, MILD PANHYPOPLASIA WITH DYSPOIESIS, SUGGESTIVE OF TOXIC MYELOPATHY, SEE COMMENT.

COMMENT: Sections of bone marrow biopsy reveal a generous segment of marrow showing 30% cellularity. There are moderate artifactual and degenerative changes of the marrow presumably due to prolonged marrow storage in RPMI solution. All cellular components appear represented but there is a moderate shift to the left with moderate dyspoietic features of all three cell lines. Occasional small aggregates of lymphoid cells are noted which appear to have atypical morphology. Also noted scattered within the marrow are increased numbers of histiocytic cells. There also appears to be a focal increase in reticular fibrosis. No blastic infiltrates, or granulomata are noted. Bony trabeculae appear unremarkable. Stainable iron stores are essentially absent.

The histologic features are somewhat obscured by artifacts induced by prolonged storage in RPMI solution. The overall left shift in maturation, dyspoietic features, and hypocellularity suggest the possibility of a toxic myelopathy. No evidence for acute blastic leukemia or aplastic anemia is present. A repeat bone marrow biopsy and bone marrow aspiration for flow cytometric immunophenotypic analysis is recommended for more definitive evaluation if pancytopenia persists. See also Flow Cytometry immunophenotypic analysis report F98-0460.

GROSS: A. Received is a cylindrical fragment of firm tissue measuring 3.3 x 0.2 x 0.2 cm. The specimen is submitted entirely in 1 cassette. DG/NSN/adb

[redacted]

jki

500 Gypsy Lane · Youngstown, Ohio 44501 · Phone (330) 740-3767 · Fax (330) 740-3790
WESTERN RESERVE CARE SYSTEM

Forum HEALTH

NORTHSIDE MEDICAL CENTER
TOD CHILDREN'S HOSPITAL
BEEGHLY MEDICAL PARK

DEPARTMENT OF LABORATORY MEDICINE

1

SURGICAL PATHOLOGY REPORT

PATIENT: [redacted]
ADDRESS: [redacted]
AGE/SEX: 9Y M
BIRTHDATE: 05/31/1989
SOC SEC: [redacted]

ACCESS NO: S98-13639
HOSP NO: 0272192
ACCOUNT NO: 002721920029
PAT LOC: T307B (TOD 3)
CPT CODE: 88305 X4/88311 X4

SURGEON: [redacted]
RESIDENT:
ATTEND PHY: [redacted]
COPY TO PHY:

DATE OF OPERATION: 10/20/98
DATE OF ACCESSION: 10/20/98
DATE OF REPORT: 10/22/98

PRE OP DX: PANCYTOPENIA
OPERATION: BILATERAL BONE MARROW ASPIRATE AND BX.
POST OP DX: SAME --ADB
HISTORY:

PREV ACC: F98-0476 S98-13470 F98-0460 S98-13175 X98-3088 S94-07494 S89-09860

TISSUES REMOVED: A: RT. ASIS
B: LT. ASIS
C: RT. PSIS
D: LT. PSIS

INTRA/EXTRA CONSULT: INTRA/EXTRA [redacted] UNIVERSITY OF NEW MEXICO SCHOOL OF MEDICINE)

==========

FINAL DIAGNOSIS:

A. RIGHT ANTERIOR BONE MARROW BIOPSY, DYSPOIETIC BONE MARROW SUGGESTIVE OF MYELODYSPLASTIC SYNDROME (SEE COMMENT).
B. LEFT ANTERIOR BONE MARROW BIOPSY, DYSPOIETIC BONE MARROW SUGGESTIVE OF MYELODYSPLASTIC SYNDROME (SEE COMMENT).
C. RIGHT POSTERIOR BONE MARROW BIOPSY, DYSPOIETIC BONE MARROW SUGGESTIVE OF MYELODYSPLASTIC SYNDROME (SEE COMMENT).
D. LEFT POSTERIOR BONE MARROW BIOPSY, DYSPOIETIC BONE MARROW SUGGESTIVE OF MYELODYSPLASTIC SYNDROME (SEE COMMENT).

COMMENT: The bone marrow biopsies (A,B,C,D) show similar histologic features. The biopsies are normocellular to mildly hypercellular for age (80 to 90% cellularity). Megakaryocytes are present and showed dyspoietic morphology. Micromegakaryocytes and uninucleate megakaryocytes are identified. Rare foci of emperipolesis is also noted. Myeloid and erythroid precursors are present and show dyspoietic maturation. The myeloid series show near maturation arrest with only scattered mature myeloid cells present. Blasts appear increased and comprise approximately 10 to 15% of nucleated marrow cells. Erythroid precursors appear somewhat decreased and show mild dyspoietic features. On PAS stained sections, the M:E ratio is approximately 4:1. Mildly increased number of histocytes are present in the bone marrow.

HEMATOPATHOLOGY REPORTS

UNM Department of Pathology/
Tricore Reference Laboratories
2211 Lomas Blvd NE
Albuquerque, NM 87100

Director: [redacted]

Patient Name: [redacted]
Medical Record #: (00000)004256776
DOB: 05/31/1989 **Age:** 9 YRS **Sex:** M
Account Number: 0111067435
Ordered by: [redacted]
Accession No.: HR-98-001169
Date Collected: 10/23/98

HEMATOPATHOLOGY

Diagnosis:

PERIPHERAL BLOOD:
PANCYTOPENIA WITH DYSPOIETIC CHANGES AND RARE BLASTS
BONE MARROW TOUCH PREPARATION AND BIOPSIES:
MALIGNANT BONE MARROW INFILTRATE (SEE COMMENT)

Dictated by: [redacted]

Reported: 11/02/98 Electronic Signature(s)

HLE:CL :CL

Comment:

The extensive marrow infiltrate of large cells is morphologically malignant and is not compatible with a reactive process. The presence of dyspoietic changes in the peripheral blood suggests that the marrow malignancy likely represents a myeloid process (such as high grade myelodysplasia). However, we were unable to characterize the phenotype of the large immature appearing cells by immunoperoxidase staining.

To better characterize this disorder a repeat bone marrow examination with further material for special studies would be helpful. If a bone marrow aspirate can be obtained, fresh material sent for flow cytometric and cytogenetic studies as well as morphology would be useful. If an aspirate cannot be obtained, extra touch preps should be made for both cytochemical stains and immunohistochemistry. In addition, an unfixed bone marrow core biopsy would be useful (fresh cells can sometimes be recovered from biopsies for flow/cytogenetic studies). We shall be glad to provide any further assistance on the optimal method of transporting/processing these specimens.

This case was reviewed and discussed with Dr. Kathy Foucar MD who concurs with the interpretation.

Referral Accession Number:
Received: 1 slide labeled "98-62-IP", 2 slides labeled "98-63-IP", 5 slides labeled "S98-13639" and 1 block labeled "N13639-B".

Returned: 8 slides and 2 block.

Clinical Data:
The patient is a previously healthy nine-year old male. He presents with a two-month history of feeling rundown. A complete blood count revealed pancytopenia. Peripheral blood parameters on 10-26-98 reported as follows at the referring institution:

Copy To: [redacted]

UNM Department of Pathology/
Tricore Reference Laboratories
2211 Lomas Blvd NE
Albuquerque, NM 87106

Director: [redacted]

Patient Name: [redacted]
Medical Record #: (00000)004256776
DOB: 05/31/1989 **Age:** 9 YRS **Sex:** M
Account Number: 0111067435
Ordered by: [redacted]
Accession No.: HR-98-001169
Date Collected: 10/23/98

HEMATOPATHOLOGY

Clinical Data:

Peripheral blood smear:
WBC: 1.5 X 10E3/mm3
RBC: 3.1 X 10E6/mm3
Hgb: 9.4 g/dl
Hct: 27 %
Plts: 63 X 10E3/mm3

MCV: 85 fl

RDW-CV: 13 %

Neut: 3.3 %
Lymph: 75 %
Mono: 15 %
Eo: 0 %
Baso: 6 %

Morphology:

Peripheral Blood Smear:
The blood smear shows pancycpenia. Red cells show moderate anisopoikilocytosis. Platelets include large and hypogranular forms. Neutrophils are markedly reduced but show normal granulation. Rare blasts are identified.

Bone Marrow Aspirate/Touch Prep-Clot and Biopsy:

Slides depict touch preps and bone marrow core biopsy. Touch preps are paucicellular but show numerous large, atypical hematopoietic cells with fine and delicate chromatin. Maturing hematopoietic elements are also seen.

The bone marrow core biopsy shows variable cellularity, overall approximately 80% cellular. Megakaryocytes are easily identified but in many cases are small and somewhat atypical. The marrow is involved by a diffuse interstitial infiltrate of large, abnormal hematopoietic-appearing cells. These cells show round to slightly irregular nuclear contours, vesicular chromatin, occasional nucleoli and scant to moderately abundant cytoplasm. The erythroid lineage is decreased. Multiple large lymphoid aggregates are identified.

Immunophenotype:

Immunohistochemical stains performed on paraffin-embedded tissue at the University of New Mexico reveal the following: myeloperoxidase stains occasional mononuclear cells, mainly with morphology suggestive of promyelocytes or myelocytes. There is rare, equivocal staining of the abnormal cells. CD34 stains blood vessels and a small minority of the abnormal cells. CD3 and CD20 stain lymphocytes. Hemoglobin A stains rare, scattered islands of erythroid precursors.

Reviewed by: [redacted]

Copy To: [redacted]

Report Date/Time: 11/02/98 1602
Continued . . .

UNM Department of Pathology/
Tricore Reference Laboratories
2211 Lomas Blvd NE
Albuquerque, NM 87106

[redacted]

Patient Name:
Medical Record #: (00000)004256776
DOB: 05/31/1989 **Age:** 9 YRS **Sex:** M
Account Number: 0111067435
Ordered by: [redacted]
Accession No.: HR-98-001169
Date Collected: 10/23/98

HEMATOPATHOLOGY

Referral MD:
[redacted]
Forum Health
500 Gypsy Lane
Box 240
Youngstown, OH 44501-0240

Physician Review/Verification:
With the exception of "Banked only" specimens, this diagnosis is based on the staff pathologist's review of the report, all microscope slides, and (if performed) flow cytometric studies and electron microscopic images.

Copy To: [redacted]

Report Date/Time: 11/02/98 1602
End of Report

APPENDIX 2

BLOOD VALUES

DISPLAY RESULTS RESULTS DISPLAY 808

M 9 TOD3 T314A PDM PT NO: 2721920045

ADM DT: 11/18/98 PT STS: IA ISOL: I MR NO: 00272192

---PAGE 1 DOWN, ACROSS 1 OF 1

	?1		?2	?3		?4
BLOOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
? WBC	0.4	L	(5.0-14.5)	TH/CMM	11/30/98	05:00
CRITICAL VALUE PHONED TO*MR AT 0725 BY NMB						
? RBC	2.43	L	(4.00-5.20)	MILL/C	11/30/98	05:00
? HEMOGLOBIN	7.2	L	(11.5-15.5)	G%	11/30/98	05:00
? HEMATOCRIT	20.3	L	(35.0-45.0)	%	11/30/98	05:00
? MCV	83.6		(77-95)	CMU	11/30/98	05:00
? MCH	29.6		(25-33)	MCGM	11/30/98	05:00
? MCHC	35.4		(31-37)	%	11/30/98	05:00
? RDW	12.7		(11.5-14.5)	%	11/30/98	05:00
? WBC DIFF					11/30/98	05:00
? WBC MORPH					11/30/98	05:00

WBC DECREASED; NO DIFFERENTIAL PERFORMED

---------------------- GROUP CONTINUED ON NEXT PAGE ----------------------

! (PF14) PATIENT MENU !(PF5) DETAIL

!(PF9) SAVE

(PF17) PRINT ALL SCREENS ! PF8 DOWN !(PF12) GRAPH

! (PF16) DISPLAY MENU

REDRTG01

DISPLAY RESULTS RESULTS DISPLAY 808

9 TOD3 T314A PDM PT NO: 2721920045

ADM DT: 11/18/98 PT STS: IA ISOL: I MR NO: 00272192

---PAGE 2 DOWN, ACROSS 1 OF 1

		?1	?2		?3		?4
	BLOOD COUNT CONT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
?	PLT COUNT	34	L	(140-440)	TH/CMM	11/30/98	05:00

	CHEM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
?	CL	102		(101-111)	MMOL/L	11/30/98	05:00
?	K	3.4	L	(3.5-5.0)	MMOL/L	11/30/98	05:00
?	NA	137		(136-145)	MMOL/L	11/30/98	05:00
?	BUN	5	L	(6-19)	MG/DL	11/30/98	05:00
?	GLUCOSE,TM	107		(70-110)	MG/DL	11/30/98	05:00
?	PROTEIN,TOT.	5.1	L	(6.0-8.5)	GM/DL	11/30/98	05:00
?	ALBUMIN	2.8	L	(3.9-4.8)	GM/DL	11/30/98	05:00
?	CA	8.3	L	(8.5-10.5)	MG/DL	11/30/98	05:00
?	CREATININE	0.3	L	(0.5-1.1)	MG/DL	11/30/98	05:00

---------------------- GROUP CONTINUED ON NEXT PAGE ----------------------

! (PF14) PATIENT MENU ! PF6 MAX UP !(PF5) DETAIL

! PF7 UP !(PF9) SAVE

(PF17) PRINT ALL SCREENS ! PF8 DOWN !(PF12) GRAPH

! (PF16) DISPLAY MENU

REDRTG01

```
DISPLAY RESULTS                          RESULTS DISPLAY                          809
                              M    9   TOD3   T314A   PDM   PT NO:   2721920045
ADM DT: 11/18/98   PT STS:    IA                    ISOL: I   MR NO: 00272192
-------------------------------------------------PAGE   3 DOWN, ACROSS   1 OF   1
                          ?1             ?2             ?3              ?4
   CHEM PROFILE  CONT     VALUE     ABN    NORMAL RANGE     UNIT   DATE    TIME
?    BILI,TOTAL           0.6             (0.1-1.5)        MG/DL  11/30/98 05:00
?    ALK PHOS             116       L     (117-390)        U/L    11/30/98 05:00
?    SGOT                 17              (0-37)           U/L    11/30/98 05:00
?    TRIGLYCERIDE         119             (<200)           MG/DL  11/30/98 05:00
?    CO2                  26              (23-29)          MMOL/L 11/30/98 05:00
   ------------------------------------------------------------------------------
   CHEMISTRIES            VALUE     ABN    NORMAL RANGE     UNIT   DATE    TIME
?    MAGNESIUM            1.6       L     (1.7-2.2)        MG/DL  11/30/98 05:00


   ------------------------------ END OF DISPLAY ----------------------------
 ! (PF14) PATIENT MENU          ! PF6 MAX UP                        !(PF5) DETAIL
                                ! PF7 UP                            !(PF9) SAVE
 (PF17) PRINT ALL SCREENS                                          !(PF12) GRAPH
 ! (PF16) DISPLAY MENU
REDRTG01
```

	12/01/98	11/30/98
BLOOD COUNT	05:00	05:00
WBC	0.4*	0.4*
RBC	2.28*	2.43*
HEMOGLOBIN	6.7*	7.2*
HEMATOCRIT	18.9*	20.3*
MCV	82.7	83.6
MCH	29.5	29.6
MCHC	35.6	35.4
RDW	12.3	12.7
WBC DIFF		
WBC MORPH	SEE TEXT	SEE TEXT
PLT COUNT	49*	34*

B

	12/01/98	11/30/98
CHEM PROFILE	05:00	05:00
CL	102	102
CO2	26	26
K	3.3*	3.4*
NA	135*	137
ANION GAP	10	
BUN	3*	3*
GLUCOSE,FM	96	107

AB

NB

==========GROUP CONTINUED==========

08:31 12/01/98 FROM WKJ3,REPRTOP1
32W87075

RESULTS LISTING
2721920045 TOD3 T314A PDM
MR# 00272192

	12/01/98	11/30/98
CHEM PROFILE CON	[illegible]	05:00
PROTEIN,TOT.		5.1*
ALBUMIN		2.6*
CA	7.9*	8.3*
CREATININE	0.3*	0.3*
BILI,TOTAL		0.6
ALK PHOS		116*
SGOT		17
TRIGLYCERIDE		119

	12/01/98	11/30/98		
CHEMISTRIES	05:00	23:00	21:00	05:00
MAGNESIUM	1.7			1.6
VANCOMYCIN			5.2	
VANCOMYCIN,PK		27.4		

BLOOD BANK SPECIMEN NO. [illegible]-1 DATE/TIME: 12/01/98 [illegible]
COMPONENT 12/01/98 01:40
PHERES PLAT LEDEP
UNIT NUMBER 12/01/98 01:40
42FR74094PHPK
==========BLOOD BANK/MICRO RESULTS CONTINUED ON NEXT PAGE==========

08:31 12/01/98 FROM WKJ3,REPRTOP1
32W87075

RESULTS LISTING
2721920045 [redacted] TOD3 T314A PDM
MR# 00272192

BLOOD BANK CONTINUED SPECIMEN NO. [illegible] DATE/TIME: [illegible]
UNIT STATUS 12/01/98 [illegible]
ALLOCATED
TRANSFUSED 12/01/98 [illegible]
OK TO TRANSFUSE

NB

RESULTS LISTING

MR# 00272192

BLOOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE
WBC	6.4	L	(7.0-16.3)	TH/CMM	12/04/98
RBC	3.99	L	(4.00-5.20)	MILL/C	12/04/98
HEMOGLOBIN	7.3	L	(11.5-15.5)	GX	12/04/98
HEMATOCRIT	20.8	L	(33.0-45.0)	%	12/04/98
MCV	61.5		(72-95)	CUL	12/04/98
MCH	29.0		(25-33)	MCGM	12/04/98
MCHC	35.5		(31-37)	%	12/04/98
RDW	12.2		(11.5-14.5)	%	12/04/98
WBC DIFF					12/04/98
WBC MORPH					12/04/98

WBC DECREASED; NO DIFFERENTIAL PERFORMED

RBC MORPH	NORMAL				12/04/98
PLT COUNT	30	L	(140-440)	TH/CMM	12/04/98

CHEM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE
CL	99	L	(101-111)	MMOL/L	12/04/98
CO2	27		(23-29)	MMOL	12/04/98
K	4.1		(3.5-5.0)	MMOL/L	12/04/98
NA	133	L	(136-145)	MMOL/L	12/04/98
ANION GAP	5		(7-20)		12/04/98
BUN	7	L	(8-17)	MG/DL	12/04/98
GLUCOSE,FM	104	H	(70-110)	MG/DL	12/04/98

104.

CONTINUED

RESULTS LISTING

MR# 00272192

CHEM PROFILE (CONT)	VALUE	ABN	NORMAL RANGE	UNIT	DATE
CREATININE	1.0		(0.5-1.1)	MG/DL	12/04/98
UA	5.5		(3.0-10.5)	MG/DL	12/04/98
ALBUMIN	3.1	L	(3.7-4.6)	GM/DL	12/04/98
BILI,TOTAL	1.5		(0.1-1.0)	MG/DL	12/04/98
BILI,DIRECT	0.1		(0.0-0.4)	MG/DL	12/04/98
ALK PHOS	103		(30-120)	U/L	12/04/98
SGPT	15		(5-50)	U/L	12/04/98
SGOT	17		(5-50)	U/L	12/04/98

URINALYSIS	VALUE	ABN	NORMAL RANGE	UNIT	DATE
PROTEIN,QUA	1.5		(1.0-2.0)	MG/DL	12/04/98

* Another?

BLOOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
WBC	0.4	L	(5.0-14.5)	TH/CMM	12/05/98	05:0
CRITICAL VALUE PHONED TO*CF AT 0807 BY VR						
RBC	2.28	L	(4.00-5.20)	MILL/C	12/05/98	05:0
HEMOGLOBIN	6.8	L	(11.5-15.5)	G%	12/05/98	05:0
HEMATOCRIT	19.2	L	(35.0-45.0)	%	12/05/98	05:0
MCV	83.9		(77-95)	CMU	12/05/98	05:0
MCH	29.6		(25-33)	MCGM	12/05/98	05:0
MCHC	35.3		(31-37)	%	12/05/98	05:0
RDW	12.0		(11.5-14.5)	%	12/05/98	05:0
WBC DIFF					12/05/98	05:0
WBC MORPH					12/05/98	05:0
WBC DECREASED; NO DIFFERENTIAL PERFORMED						
PLT COUNT	23	L	(140-440)	TH/CMM	12/05/98	05:0
CRITICAL VALUE PHONED TO*CF AT 0807 BY VR						

CHEM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
CL	106		(101-111)	MMOL/L	12/05/98	05:0
CO2	26		(23-29)	MMOL/L	12/05/98	05:0
K	4.2		(3.5-5.0)	MMOL/L	12/05/98	05:0
NA	140		(136-145)	MMOL/L	12/05/98	05:0
ANION GAP	12		(10-20)		12/05/98	05:0
BUN	10		(6-19)	MG/DL	12/05/98	05:0

=========GROUP CONTINUED=========

.3:22 12/05/98 FROM WKH5,REPRTGF1
JAX35017

RESULTS LISTING
272192004[illegible] TOD3 T305B PDM
MR# 002/2192

CHEM PROFILE CONT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
GLUCOSE,TM	102		(70-110)	MG/DL	12/05/98	05:0
CREATININE	0.3	L	(0.5-1.1)	MG/DL	12/05/98	05:0
CA	8.9		(8.5-10.5)	MG/DL	12/05/98	05:0

CHEMISTRIES	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
MAGNESIUM	2.1		(1.7-2.2)	MG/DL	12/05/98	05:0

URINALYSIS	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
U PH	5.0		(5.0-8.0)		12/05/98	02:0
U SP GRAVIT	1.010		(1.030)		12/05/98	02:0

BLOOD BANK	SPECIMEN NO: 206-1-1	DATE/TIME: 12/05/98 05:00
ABO/RH	12/05/98 05:00	
B*POS		
ANTIBDY SC	12/05/98 05:00	
NEG		
COMPONENT	12/05/98 05:00	
RBC-A IRAD		
UNIT NUMBER	12/05/98 05:00	
42FX76787		

=========BLOOD BANK/MICRO RESULTS CONTINUED ON NEXT PAGE=========

L3:22 12/05/98 FROM WKH5,REPRTGF1
JAX35017

RESULTS LISTING
272192004[illegible] TOD3 T305B PDM
MR# 002/2192

BLOOD BANK CONTINUED	SPECIMEN NO: 206-1-1	DATE/TIME: 12/05/98 05:00
UNIT STATUS	12/05/98 05:00	
ALLOCATED		
TRNSF.STATU	12/05/98 05:00	
OK TO TRANSFUSE		
CROSSMATCH	12/05/98 05:00	
COMPATIBLE		

BLOOD COUNT	[illegible]	12/06/98 05:00	[illegible]
WBC	[illegible]	0.3*	[illegible]
RBC	2.86*	3.06*	2.28*
HEMOGLOBIN	[illegible]	8.7*	6.8*
HEMATOCRIT	23.7*	25.0*	19.2*
MCV	82.8	81.5	83.7
MCH	29.3	29.0	29.6
MCHC	35.3	35.5	35.3
RDW	11.9	12.3	12.0
WBC DIFF			
WBC MORPH	SEE TEXT	SEE TEXT	SEE TEXT
PLT COUNT	[illegible]	28*	39*

CHEM PROFILE	12/07/98 05:00	12/06/98 05:00	12/03/98 05:00
CL	102	98*	106
CO2	[illegible]	27	26
K	4.1	4.1	4.2
NA	[illegible]	134*	140
ANION GAP		13	12
BUN	12	11	10
GLUCOSE,TM	114*	110	102

GROUP CONTINUED

08:40 12/07/98 FROM WAAL,REPRTGP1
52WB7628

RESULTS LISTING
272192004

MR# 00272192

TOD3 T3056 POM

103.9°
Vanco
Fortaz
Diflucan
TPN

G-CSF

CHEM PROFILE CON	12/07/98 05:00	12/06/98 05:00	12/03/98 05:00
[illegible]	3.1		
ALBUMIN	3.4*		
CA	8.7	8.4*	[illegible]
CREATININE	[illegible]	0.3*	0.3*
BILI,TOTAL	0.7		
ALK PHOS	107*		
SGOT	24		

CHEMISTRIES	12/07/98 05:00	12/06/98 05:00	12/03/98 05:00
MAGNESIUM	[illegible]	[illegible]	[illegible]

URINALYSIS	[illegible]	[illegible]
URINALYSIS		
[illegible]	[illegible]	
U APPEARANCE	SEE TEXT	
SP GRAVITY	[illegible]	[illegible]
U PH	[illegible]	
U PROTEIN	[illegible]	
[illegible]	[illegible]	

[illegible]

RESULTS LISTING
[illegible]

TOD3 T3056 POM

[illegible]

[illegible]

08:41 12/09/98 FROM ;+- ,REERPRF6
32WB7964

RESULTS LISTING
2721920045 TOD3 T305B PDM
MR# 00272192

BLOOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TI
WBC	0.5	L	(5.0-14.5)	TH/CMM	12/09/98	05
CRITICAL VALUE PHONED TO*KC T3 AT 0615 BY AS						
RBC	2.63	L	(4.00-5.20)	MILL/C	12/09/98	05
HEMOGLOBIN	7.6	L	(11.5-15.5)	G%	12/09/98	05
HEMATOCRIT	21.6	L	(35.0-45.0)	%	12/09/98	05
MCV	82.2		(77-95)	CMU	12/09/98	05
MCH	28.9		(25-33)	MCGM	12/09/98	05
MCHC	35.2		(31-37)	%	12/09/98	05
RDW	11.7		(11.5-14.5)	%	12/09/98	05
WBC DIFF					12/09/98	05
LYMPHOCYTE	96	H	(20-44)	%	12/09/98	05
ATYP/REACT	4			%	12/09/98	05
RBC MORPH	NORMAL				12/09/98	05
PLT COUNT	36	L	(140-440)	TH/CMM	12/09/98	05

CHEM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TI
CL	105		(101-111)	MMOL/L	12/09/98	05
CO2	26		(23-29)	MMOL/L	12/09/98	05
K	4.3		(3.5-5.0)	MMOL/L	12/09/98	05
NA	140		(136-145)	MMOL/L	12/09/98	05
ANION GAP	13		(10-20)		12/09/98	05
BUN	16		(6-19)	MG/DL	12/09/98	05

=GROUP CONTINUED=

09:01 12/09/98 FROM WKJ3,REPRTGP1
32WB7965

RESULTS LISTING
2721920045 TOD3 T305B PDM
MR# 00272192

CHEM PROFILE CONT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TI
GLUCOSE,TM	142	H	(70-110)	MG/DL	12/09/98	05
CREATININE	0.3	L	(0.5-1.1)	MG/DL	12/09/98	05
CA	7.4		(8.5-10.5)	MG/DL	12/09/98	05

CHEMISTRIES	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TI
MAGNESIUM	2.3	H	(1.7-2.2)	MG/DL	12/09/98	05

BLOOD BANK SPECIMEN NO: 294-1-1 DATE/TIME: 12/09/98 00:
COMPONENT 12/09/98 00:10
FRENES FLAT LEDEP
UNIT NUMBER 12/09/98 00:10
*2FR24195PHFR
UNIT STATUS 12/09/98 00:10
ALLOCATED
TRNSF.STATU 12/09/98 00:10
OK TO TRANSFUSE

RESULTS LISTING
2721920045 MR# 00272192 10D3 T305B PDM

BLOOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TI
WBC	0.3	L	(5.0-14.5)	TH/CMM	12/11/98	05
CRITICAL VALUE PHONED TO*CM AT 0640 BY JAB						
RBC	2.63	L	(4.00-5.20)	MILL/C	12/11/98	05
HEMOGLOBIN	7.9	L	(11.5-15.5)	G%	12/11/98	05
HEMATOCRIT	22.2	L	(35.0-45.0)	%	12/11/98	05
MCV	84.3		(77-95)	CMU	12/11/98	05
MCH	29.9		(25-33)	MCGM	12/11/98	05
MCHC	35.4		(31-37)	%	12/11/98	05
RDW	12.5		(11.5-14.5)	%	12/11/98	05
PLT COUNT	20	L	(140-440)	TH/CMM	12/11/98	05
CRITICAL VALUE PHONED TO*CM AT 0640 BY JAB						

CHEM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TI
CL	105		(101-111)	MMOL/L	12/11/98	05
CO2	26		(23-29)	MMOL/L	12/11/98	05
K	4.0		(3.5-5.0)	MMOL/L	12/11/98	05
NA	139		(136-145)	MMOL/L	12/11/98	05
ANION GAP	[illegible]		(10-20)		12/11/98	05
BUN	13		(6-19)	MG/DL	12/11/98	05
GLUCOSE	[illegible]		[illegible]	[illegible]	[illegible]	[illegible]
CREATININE	0.3	L	(0.5-1.1)	MG/DL	12/11/98	05
CA	8.7		(8.5-10.5)	MG/DL	12/11/98	05

REPORT CONTINUED ON NEXT PAGE

08:25 12/11/98 FROM WKJ3,REPRTGF1
32WB8356

RESULTS LISTING
2721920045 MR# 00272192 10D3 T305B PDM

CHEMISTRIES	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TI
MAGNESIUM	2.0		(1.7-2.2)	MG/DL	12/11/98	05

BM pending

END OF REPORT

08:25 12/11/98 FROM WKJ3,REPRTGF1
32WB8356

RESULTS LISTING
2721920045 TOD3 T305B PDM
MR# 00272192

BLOOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIM
WBC	0.2	L	(5.0-14.5)	TH/CMM	12/12/98	05:0
CRITICAL VALUE PHONED TO*I3 AT 0610 BY AS						
RBC	2.62	L	(4.00-5.20)	MILL/C	12/12/98	05:0
HEMOGLOBIN	7.7	L	(11.5-15.5)	G%	12/12/98	05:0
HEMATOCRIT	21.9	L	(35.0-45.0)	%	12/12/98	05:0
MCV	83.7		(77-95)	CMU	12/12/98	05:0
MCH	29.4		(25-33)	MCGM	12/12/98	05:0
MCHC	35.1		(31-37)	%	12/12/98	05:0
RDW	12.4		(11.5-14.5)	%	12/12/98	05:0
WBC DIFF					12/12/98	05:0
WBC MORPH					12/12/98	05:0
WBC DECREASED; NO DIFFERENTIAL PERFORMED						
RBC MORPH	NORMAL				12/12/98	05:0
PLT COUNT	45	L	(140-440)	TH/CMM	12/12/98	05:0

CHEM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
CL	101		(101-111)	MMOL/L	12/12/98	05:0
CO2	25		(23-29)	MMOL/L	12/12/98	05:0
K	4.4		(3.5-5.0)	MMOL/L	12/12/98	05:0
NA	136		(136-145)	MMOL/L	12/12/98	05:0
ANION GAP	14		(10-20)		12/12/98	05:0
BUN	14		(6-19)	MG/DL	12/12/98	05:0

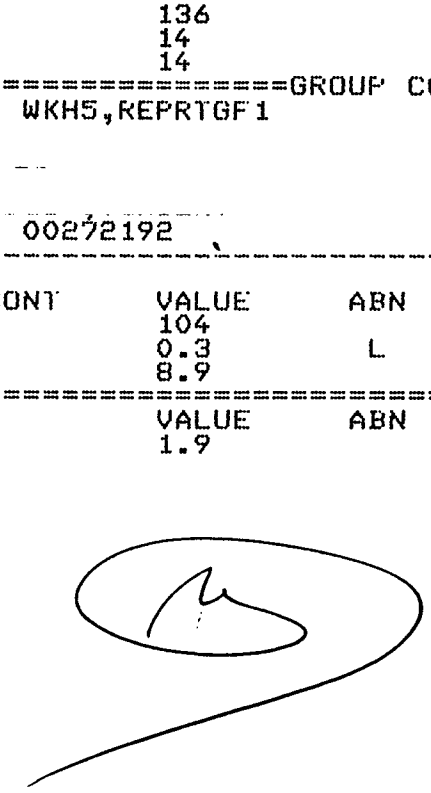
GROUP CONTINUED

09:55 12/12/98 FROM WKH5,REPRTGF1
WAX36212

RESULTS LISTING
2721920045 T305B PDM
MR# 00272192

CHEM PROFILE CONT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
GLUCOSE,TM	104		(70-110)	MG/DL	12/12/98	05:0
CREATININE	0.3	L	(0.5-1.1)	MG/DL	12/12/98	05:0
CA	8.9		(8.5-10.5)	MG/DL	12/12/98	05:0

CHEMISTRIES	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
MAGNESIUM	1.9		(1.7-2.2)	MG/DL	12/12/98	05:0



END OF REPORT

09:55 12/12/98 FROM WKH5,REPRTGF1
WAX36212

RESULTS LISTING
2721920045 TOD3 T305B PDM
MR# 00272192

BLOOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
WBC	0.3	L	(5.0-14.5)	TH/CMM	12/15/98	05:
CRITICAL VALUE PHONED TO*JE AT 0700 BY BRM						
RBC	2.90	L	(4.00-5.20)	MILL/C	12/15/98	05:
HEMOGLOBIN	8.8	L	(11.5-15.5)	G%	12/15/98	05:
HEMATOCRIT	24.6	L	(35.0-45.0)	%	12/15/98	05:
MCV	84.7		(77-95)	CMU	12/15/98	05:
MCH	30.2		(25-33)	MCGM	12/15/98	05:
MCHC	35.6		(31-37)	%	12/15/98	05:
RDW	11.8		(11.5-14.5)	%	12/15/98	05:
WBC DIFF					12/15/98	05:
WBC MORPH					12/15/98	05:
WBC DECREASED; NO DIFFERENTIAL PERFORMED						
PLT COUNT	32	L	(140-440)	TH/CMM	12/15/98	05:
WBC	0.3	L	(5.0-14.5)	TH/CMM	12/14/98	05:
CRITICAL VALUE PHONED TO*CS AT 0745 BY JK						
RBC	2.47	L	(4.00-5.20)	MILL/C	12/14/98	05:
HEMOGLOBIN	7.4	L	(11.5-15.5)	G%	12/14/98	05:
HEMATOCRIT	20.6	L	(35.0-45.0)	%	12/14/98	05:
MCV	83.5		(77-95)	CMU	12/14/98	05:
MCH	29.9		(25-33)	MCGM	12/14/98	05:
MCHC	35.7		(31-37)	%	12/14/98	05:
RDW	12.1		(11.5-14.5)	%	12/14/98	05:

=========GROUP CONTINUED=========

14:18 12/15/98 FROM WKH5,REPRTGF1
WAX36705

RESULTS LISTING
2721920045 TOD3 T305B PDM
MR# 00272192

BLOOD COUNT CONT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
WBC DIFF					12/14/98	05:
WBC MORPH					12/14/98	05:
WBC DECREASED; NO DIFFERENTIAL PERFORMED						
PLT COUNT	12	L	(140-440)	TH/CMM	12/14/98	05:
CRITICAL VALUE PHONED TO*CS AT 0745 BY JK						

CHEM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
CL	104		(101-111)	MMOL/L	12/15/98	05:
CO2	24		(23-29)	MMOL/L	12/15/98	05:
K	4.0		(3.5-5.0)	MMOL/L	12/15/98	05:
NA	138		(136-145)	MMOL/L	12/15/98	05:
ANION GAP	14		(10-20)		12/15/98	05:
BUN	15		(6-19)	MG/DL	12/15/98	05:
GLUCOSE,TM	100		(70-110)	MG/DL	12/15/98	05:
CREATININE	0.3	L	(0.5-1.1)	MG/DL	12/15/98	05:
CA	8.8		(8.5-10.5)	MG/DL	12/15/98	05:
ALBUMIN	3.4	L	(3.9-4.8)	GM/DL	12/15/98	05:
BILI,TOTAL	3.7	H	(0.1-1.5)	MG/DL	12/15/98	05:
BILI,DIRECT	2.6	H	(0-0.4)	MG/DL	12/15/98	05:
ALK PHOS	169		(117-390)	U/L	12/15/98	05:
SGPT	860	H	(0-45)	U/L	12/15/98	05:
SGOT	352	H	(0-37)	U/L	12/15/98	05:

=========GROUP CONTINUED=========

14:18 12/15/98 FROM WKH5,REPRTGF1
WAX36705

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CHEM PROFILE  CONT        VALUE      ABN  NORMAL RANGE      UNIT    DATE      TIME
  CL                      102             (101-111)         MMOL/L  12/14/98  05:0
  K                       4.3             (3.5-5.0)         MMOL/L  12/14/98  05:0
  NA                      136             (136-145)         MMOL/L  12/14/98  05:0
  BUN                     12              (6-19)            MG/DL   12/14/98  05:0
  GLUCOSE,TM              111          H  (70-110)          MG/DL   12/14/98  05:0
  PROTEIN,TOT             5.9          L  (6.0-8.5)         GM/DL   12/14/98  05:0
  ALBUMIN                 3.3          L  (3.9-4.8)         GM/DL   12/14/98  05:0
  CA                      8.8             (8.5-10.5)        MG/DL   12/14/98  05:0
  CREATININE              0.3          L  (0.5-1.1)         MG/DL   12/14/98  05:0
  BILI,TOTAL              1.9          H  (0.1-1.5)         MG/DL   12/14/98  05:0
  ALK PHOS                188             (117-390)         U/L     12/14/98  05:0
  SGOT                    285          H  (0-37)            U/L     12/14/98  05:0
  CO2                     25              (23-29)           MMOL/L  12/14/98  05:0
==========================================================================================
CHEMISTRIES               VALUE      ABN  NORMAL RANGE      UNIT    DATE      TIME
  MAGNESIUM               1.9             (1.7-2.2)         MG/DL   12/15/98  05:1
  MAGNESIUM               1.9             (1.7-2.2)         MG/DL   12/14/98  05:0
==========================================================================================
IMMUNOLOGY                VALUE      ABN  NORMAL RANGE      UNIT    DATE      TIME
  TOXO IGG                           N  (NEG)             IU/ML   12/14/98  05:0
                                EQUIVOCAL
                          A TWO FOLD CHANGE IN IU/ML BETWEEN ACUTE AND
==========================TEXT CONTINUED ON NEXT PAGE==========================
14:18 12/15/98 FROM WKH5,REPRTGF1
JAX36705

 RESULTS LISTING
   2721920045                                    JD3      T305B      PDM
              MR# 00272192
------------------------------------------------------------------------------------------

IMMUNOLOGY     CONT       VALUE      ABN  NORMAL RANGE      UNIT    DATE      TIME
  TOXO IGG                           N  (NEG)             IU/ML   12/14/98  05:0
                                CONVALESCENT PAIRED SERA SHOULD BE
                                CONSIDERED EVIDENCE OF RECENT
                                SEROCONVERSION.  SEND ANOTHER SPECIMEN IN 2
                                TO 4 WEEKS IF CLINICALLY INDICATED.
  TOXO IGM                 NEGATIVE   N  (NEG)                        12/14/98  05:0
==========================================================================================
BLOOD BANK                  SPECIMEN NO: 370-1-1     DATE/TIME: 12/14/98 08:00
  COMPONENT                    12/14/98 08:00
PHER PLT LEDP IRAD
  UNIT NUMBER                  12/14/98 08:00
42FR74238PHPK
  UNIT STATUS                  12/14/98 08:00
TRANSFUSED
  TRNSF.STATU                  12/14/98 08:00
OK TO TRANSFUSE
======================END OF MICRO/BLOOD BANK REPORT======================
BLOOD BANK                  SPECIMEN NO: 371-1-1     DATE/TIME: 12/14/98 08:00
  COMPONENT                    12/14/98 08:00
PHER PLT LEDP IRAD
=========BLOOD BANK/MICRO RESULTS CONTINUED ON NEXT PAGE==========


14:18 12/15/98 FROM WKH5,REPRTGF1
JAX36705

 RESULTS LISTING
   2721920045                                    TOD3     T305B      PDM
              MR# 00272192
------------------------------------------------------------------------------------------

BLOOD BANK     CONTINUED SPECIMEN NO: 371-1-1     DATE/TIME: 12/14/98 08:00
  UNIT NUMBER                  12/14/98 08:00
42FR67191PHPK
  UNIT STATUS                  12/14/98 08:00
ALLOCATED
  TRNSF.STATU                  12/14/98 08:00
OK TO TRANSFUSE
```

RESULTS LISTING
2721920045 MR# 00272192 1003 15056 FDM

BLOOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TI
WBC	0.4	L	(5.0-14.5)	TH/CMM	12/18/98	05
CRITICAL VALUE PHONED TO*DB AT 0535 BY AI						
RBC	2.37	L	(4.00-5.20)	MILL/C	12/18/98	05
HEMOGLOBIN	7.1	L	(11.5-15.5)	G%	12/18/98	05
HEMATOCRIT	20.1	L	(35.0-45.0)	%	12/18/98	05
MCV	84.6		(77-95)	CMU	12/18/98	05
[illegible]	[illegible]		[illegible]	[illegible]	12/18/98	05
[illegible]	[illegible]		[illegible]	%	12/18/98	05
RDW	12.2		(11.5-14.5)	%	12/18/98	05

PLT ______

CHEM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TI
CL	101		(101-111)	MMOL/L	12/18/98	05
CO2	21	L	(23-29)	MMOL/L	12/18/98	05
K	3.4	L	(3.5-5.0)	MMOL/L	12/18/98	05
NA	134	L	(136-145)	MMOL/L	12/18/98	05
ANION GAP	15		(10-20)		12/18/98	05
BUN	17		(6-19)	[illegible]	12/18/98	05
GLUCOSE,RA	[illegible]	H	(70-110)	[illegible]	12/18/98	05
CREATININE	0.3	L	(0.5-1.1)	MG/DL	12/18/98	05
CA	8.4	L	(8.5-10.5)	MG/DL	12/18/98	05
[illegible]	[illegible]	H	(11-51)	[illegible]	12/18/98	05

[illegible] 12/18/98 FROM [illegible]
[illegible]

RESULTS LISTING
[illegible] MR# [illegible] [illegible] [illegible] FDM

[illegible]	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TI
MAGNESIUM	1.7		[illegible]	MG/DL	12/18/98	[illegible]

RESULTS LISTING
2721920045 TOD3 T305B PDM
MR# 00272192

BLOOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TI
WBC	2.5	L	(5.0-14.5)	TH/CMM	12/23/98	05:
RBC	2.69	L	(4.00-5.20)	MILL/C	12/23/98	05:
HEMOGLOBIN	8.1	L	(11.5-15.5)	G%	12/23/98	05:
HEMATOCRIT	22.8	L	(35.0-45.0)	%	12/23/98	05:
MCV	84.8		(77-95)	CMU	12/23/98	05:
MCH	30.0		(25-33)	MCGM	12/23/98	05:
MCHC	35.4		(31-37)	%	12/23/98	05:
RDW	12.6		(11.5-14.5)	%	12/23/98	05:
WBC DIFF					12/23/98	05:
POLY %	16	L	(50-70)	%	12/23/98	05:
STAB %	46	H	(2-6)	%	12/23/98	05:
LYMPHOCYTE	27		(20-44)	%	12/23/98	05:
MONOCYTES %	5		(2-9)	%	12/23/98	05:
METAMYELOC.	3			%	12/23/98	05:
MYELOCYTE %	3			%	12/23/98	05:
WBC MORPH					12/23/98	05:
TOXIC GRAN*MOD*DOHLE BODY						
RBC MORPH	SLT*POLY				12/23/98	05:
PLT COUNT	31	L	(140-440)	TH/CMM	12/23/98	05:

CHEM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
CL	105		(101-111)	MMOL/L	12/23/98	05:

=GROUP CONTINUED=

1:21 12/23/98 FROM WKH5,REPRTGF1
JAX38023

RESULTS LISTING
2721920045 TOD3 T305B PDM
MR# 00272192

CHEM PROFILE CONT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
CO2	30	H	(23-29)	MMOL/L	12/23/98	05:
K	2.7	L	(3.5-5.0)	MMOL/L	12/23/98	05:
CRITICAL VALUE PHONED TO*MS AT 0700 BY TK						
NA	144		(136-145)	MMOL/L	12/23/98	05:
ANION GAP	12		(10-20)		12/23/98	05:
BUN	26	H	(6-19)	MG/DL	12/23/98	05:
GLUCOSE,TM	113	H	(70-110)	MG/DL	12/23/98	05:
CREATININE	0.4	L	(0.5-1.1)	MG/DL	12/23/98	05:
CA	8.9		(8.5-10.5)	MG/DL	12/23/98	05:
ALBUMIN	3.1	L	(3.9-4.8)	GM/DL	12/23/98	05:
BILI,TOTAL	1.6	H	(0.1-1.5)	MG/DL	12/23/98	05:
BILI,DIRECT	0.6	H	(0-0.4)	MG/DL	12/23/98	05:
ALK PHOS	399	H	(117-390)	U/L	12/23/98	05:
SGPT	590	H	(0-45)	U/L	12/23/98	05:
SGOT	60	H	(0-37)	U/L	12/23/98	05:

CHEMISTRIES	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
MAGNESIUM	2.0		(1.7-2.2)	MG/DL	12/23/98	05:

=END OF REPORT=

\:21 12/23/98 FROM WKH5,REPRTGF1
X38023

RESULTS LISTING
2721920086 MR# 00272192 TOD3 T305B HEM

BLOOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
WBC	3.0	L	(5.0-14.5)	TH/CMM	12/31/98	05:00
RBC	3.22	L	(4.00-5.20)	MILL/C	12/31/98	05:00
HEMOGLOBIN	9.8	L	(11.5-15.5)	G%	12/31/98	05:00
HEMATOCRIT	27.8	L	(35.0-45.0)	%	12/31/98	05:00
MCV	86.3		(77-95)	CMU	12/31/98	05:00
MCH	30.5		(25-33)	MCGM	12/31/98	05:00
MCHC	35.3		(31-37)	%	12/31/98	05:00
RDW	12.2		(11.5-14.5)	%	12/31/98	05:00
PLT COUNT	80	L	(140-440)	TH/CMM	12/31/98	05:00

CHEM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
CL	105		(101-111)	MMOL/L	12/31/98	05:00
CO2	28		(23-29)	MMOL/L	12/31/98	05:00
K	3.6		(3.5-5.0)	MMOL/L	12/31/98	05:00
NA	142		(136-145)	MMOL/L	12/31/98	05:00
ANION GAP	13		(10-20)		12/31/98	05:00
BUN	10		(6-19)	MG/DL	12/31/98	05:00
GLUCOSE,TM	96		(70-110)	MG/DL	12/31/98	05:00
CREATININE	0.3	L	(0.5-1.1)	MG/DL	12/31/98	05:00

==========END OF REPORT==========
21 12/31/98 FROM WKJ3,REPRTGF1
80830

ULTS LISTING
721920086 MR# 00272192 TOD3 T305B HEM

BLOOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
WBC	2.2	L	(5.0-14.5)	TH/CMM	01/03/99	05:00
RBC	3.50	L	(4.00-5.20)	MILL/C	01/03/99	05:00
HEMOGLOBIN	10.6	L	(11.5-15.5)	G%	01/03/99	05:00
HEMATOCRIT	29.8	L	(35.0-45.0)	%	01/03/99	05:00
MCV	85.2		(77-95)	CMU	01/03/99	05:00
MCH	30.4		(25-33)	MCGM	01/03/99	05:00
MCHC	35.6		(31-37)	%	01/03/99	05:00
RDW	12.4		(11.5-14.5)	%	01/03/99	05:00
WBC DIFF					01/03/99	05:00
POLY %	79	H	(50-70)	%	01/03/99	05:00
STAB %	20	H	(2-6)	%	01/03/99	05:00
LYMPHOCYTE	1	L	(20-44)	%	01/03/99	05:00
WBC MORPH					01/03/99	05:00
TOXIC GRAN	NORMAL				01/03/99	05:00
RBC MORPH					01/03/99	05:00
PLT COUNT	139	L	(140-440)	TH/CMM	01/03/99	05:00

CHEM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
CL	98	L	(101-111)	MMOL/L	01/03/99	05:00
CO2	26		(23-29)	MMOL/L	01/03/99	05:00
K	4.1		(3.5-5.0)	MMOL/L	01/03/99	05:00
NA	134	L	(136-145)	MMOL/L	01/03/99	05:00

==========GROUP CONTINUED==========
:22 01/03/99 FROM WKH1,REPRTGF1
(39450

RESULTS LISTING
2721920086 [redacted] MR# 00272192 TOD3 T305B HEM

CHEM PROFILE CONT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
ANION GAP	14		(10-20)		01/03/99	05:00
BUN	8		(6-19)	MG/DL	01/03/99	05:00
GLUCOSE,TM	94		(70-110)	MG/DL	01/03/99	05:00
CREATININE	0.4	L	(0.5-1.1)	MG/DL	01/03/99	05:00

RESULTS LISTING
2721920086 TOD3 T305B HEM
MR# 00272192

BLOOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
WBC	1.6	L	(5.0-14.5)	TH/CMM	01/02/99	05:5
CRITICAL VALUE PHONED TO*JV AT 0710 BY JAB						
RBC	3.41	L	(4.00-5.20)	MILL/C	01/02/99	05:
HEMOGLOBIN	10.2	L	(11.5-15.5)	G%	01/02/99	05:
HEMATOCRIT	29.6	L	(35.0-45.0)	%	01/02/99	05:
MCV	86.7		(77-95)	CMU	01/02/99	05:
MCH	30.0		(25-33)	MCGM	01/02/99	05:
MCHC	34.5		(31-37)	%	01/02/99	05:
RDW	12.7		(11.5-14.5)	%	01/02/99	05:
WBC DIFF					01/02/99	05:
POLY %	57		(50-70)	%	01/02/99	05:
STAB %	35	H	(2-6)	%	01/02/99	05:
EOSINOPHIL	3		(0-4)	%	01/02/99	05:
BASOPHIL %	1		(0-2)	%	01/02/99	05:
LYMPHOCYTE	3	L	(20-44)	%	01/02/99	05:
METAMYELOC.	1			%	01/02/99	05:
RBC MORPH					01/02/99	05:
SLT*TEAR DROP*SLT*POLY						
PLT COUNT	125	L	(140-440)	TH/CMM	01/02/99	05:5

CHEM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
CL	99	L	(101-111)	MMOL/L	01/02/99	05:5

=GROUP CONTINUED=

)9:08 01/02/99 FROM WKH1,REPRTGF1
JAX39298

RESULTS LISTING
2721920086 TOD3 T305B HEM
MR# 00272192

CHEM PROFILE CONT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
CO2	25		(23-29)	MMOL/L	01/02/99	05:
K	3.9		(3.5-5.0)	MMOL/L	01/02/99	05:
NA	135	L	(136-145)	MMOL/L	01/02/99	05:
ANION GAP	15		(10-20)		01/02/99	05:
BUN	7		(6-19)	MG/DL	01/02/99	05:
GLUCOSE,TM	179	H	(70-110)	MG/DL	01/02/99	05:
CREATININE	0.2	L	(0.5-1.1)	MG/DL	01/02/99	05:

=END OF REPORT=

)9:08 01/02/99 FROM WKH1,REPRTGF1
JAX39298

JLTS LISTING
721920102 TOD3 T312B PDM
MR# 00272192

LOOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
WBC	0.1	L	(5.0-14.5)	TH/CMM	01/11/99	05:00
RITICAL VALUE PHONED TO*JV AT 0700 BY GKLJ						
RBC	3.49	L	(4.00-5.20)	MILL/C	01/11/99	05:00
HEMOGLOBIN	10.6	L	(11.5-15.5)	G%	01/11/99	05:00
HEMATOCRIT	30.0	L	(35.0-45.0)	%	01/11/99	05:00
MCV	85.9		(77-95)	CMU	01/11/99	05:00
MCH	30.5		(25-33)	MCGM	01/11/99	05:00
MCHC	35.5		(31-37)	%	01/11/99	05:00
RDW	13.3		(11.5-14.5)	%	01/11/99	05:00
WBC DIFF					01/11/99	05:00
WBC MORPH					01/11/99	05:00
3C DECREASED; NO DIFFERENTIAL PERFORMED						
PLT COUNT	43	L	(140-440)	TH/CMM	01/11/99	05:00

HEM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
ALBUMIN	3.6	L	(3.9-4.8)	GM/DL	01/11/99	10:30
BILI,TOTAL	1.3		(0.1-1.5)	MG/DL	01/11/99	10:30
BILI,DIRECT	0.4		(0-0.4)	MG/DL	01/11/99	10:30
ALK PHOS	179		(117-390)	U/L	01/11/99	10:30
SGPT	39		(0-45)	U/L	01/11/99	10:30
SGOT	18		(0-37)	U/L	01/11/99	10:30
CL	103		(101-111)	MMOL/L	01/11/99	05:00

=GROUP CONTINUED=

01/11/99 FROM WKH1,REPRTGF1
557

JLTS LISTING
721920102 TOD3 T312B PDM
MR# 00272192

HEM PROFILE CONT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
CO2	24		(23-29)	MMOL/L	01/11/99	05:00
K	3.1	L	(3.5-5.0)	MMOL/L	01/11/99	05:00
NA	138		(136-145)	MMOL/L	01/11/99	05:00
ANION GAP	14		(10-20)		01/11/99	05:00
BUN	7		(6-19)	MG/DL	01/11/99	05:00
GLUCOSE,TM	93		(70-110)	MG/DL	01/11/99	05:00
CREATININE	0.3	L	(0.5-1.1)	MG/DL	01/11/99	05:00

HEMISTRIES	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
VANCOMYCN,P	20.1		(5-40)	uG/ML	01/11/99	10:30
PREALBUMIN	14.6	L	(20-43)	MG/DL	01/11/99	10:30
VANCOMYCIN	4.4	L	(5-40)	MCG/ML	01/11/99	07:52

=END OF REPORT=

01/11/99 FROM WKH1,REPRTGF1
557

LTS LISTING
21920102 TOD3 T312B PDM
MR# 00272192

OOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
WBC	0.1	L	(5.0-14.5)	TH/CMM	01/18/99	05:00
1TICAL VALUE PHONED TO*DW AT 0605 BY CM						
RBC	2.65	L	(4.00-5.20)	MILL/C	01/18/99	05:00
HEMOGLOBIN	7.8	L	(11.5-15.5)	G%	01/18/99	05:00
HEMATOCRIT	22.7	L	(35.0-45.0)	%	01/18/99	05:00
MCV	85.4		(77-95)	CMU	01/18/99	05:00
MCH	29.3		(25-33)	MCGM	01/18/99	05:00
MCHC	34.3		(31-37)	%	01/18/99	05:00
RDW	11.8		(11.5-14.5)	%	01/18/99	05:00
WBC DIFF					01/18/99	05:00
WBC MORPH					01/18/99	05:00
C DECREASED; NO DIFFERENTIAL PERFORMED						
PLT COUNT	57	L	(140-440)	TH/CMM	01/18/99	05:00

EM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
CL	101		(101-111)	MMOL/L	01/18/99	05:00
CO2	28		(23-29)	MMOL/L	01/18/99	05:00
K	3.9		(3.5-5.0)	MMOL/L	01/18/99	05:00
NA	136		(136-145)	MMOL/L	01/18/99	05:00
ANION GAP	11		(10-20)		01/18/99	05:00
BUN	7		(6-19)	MG/DL	01/18/99	05:00
GLUCOSE,TM	97		(70-110)	MG/DL	01/18/99	05:00

==========GROUP CONTINUED==========

LTS LISTING
21920102 TOD3 T312B PDM
MR# 00272192

EM PROFILE CONT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
CREATININE	0.3	L	(0.5-1.1)	MG/DL	01/18/99	05:00

==========END OF REPORT==========

ESULTS LISTING
2721920102 J3 T312B FDM
MR# 00272192

BLOOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
WBC	0.1	L	(5.0-14.5)	TH/CMM	01/20/99	05:00
CRITICAL VALUE PHONED TO*KC AT 0635 BY JAB						
RBC	3.63	L	(4.00-5.20)	MILL/C	01/20/99	05:00
HEMOGLOBIN	10.9	L	(11.5-15.5)	G%	01/20/99	05:00
HEMATOCRIT	31.2	L	(35.0-45.0)	%	01/20/99	05:00
MCV	86.0		(77-95)	CMU	01/20/99	05:00
MCH	30.1		(25-33)	MCGM	01/20/99	05:00
MCHC	35.0		(31-37)	%	01/20/99	05:00
RDW	12.6		(11.5-14.5)	%	01/20/99	05:00
WBC DIFF					01/20/99	05:00
WBC MORPH					01/20/99	05:00
WBC DECREASED; NO DIFFERENTIAL PERFORMED						
PLT COUNT	33	L	(140-440)	TH/CMM	01/20/99	05:00

CHEM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
CL	98	L	(101-111)	MMOL/L	01/20/99	05:00
CO2	30	H	(23-29)	MMOL/L	01/20/99	05:00
K	4.2		(3.5-5.0)	MMOL/L	01/20/99	05:00
NA	137		(136-145)	MMOL/L	01/20/99	05:00
ANION GAP	13		(10-20)		01/20/99	05:00
BUN	11		(6-19)	MG/DL	01/20/99	05:00
GLUCOSE,TM	110		(70-110)	MG/DL	01/20/99	05:00

=GROUP CONTINUED=

14 01/20/99 FROM WKJ3,REPRTGF1
33012

ESULTS LISTING
2721920102 J3 T312B FDM
MR# 00272192

CHEM PROFILE CONT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
CREATININE	0.4	L	(0.5-1.1)	MG/DL	01/20/99	05:00

pCO2

*candida

pH 7.42

=END OF REPORT=

14 01/20/99 FROM WKJ3,REPRTGF1
33012

RESULTS LISTING
2721920102
MR# 00272192 TOD3 T312B PDM

BLOOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	
WBC	0.3	L	(5.0-14.5)	TH/CMM	01/21/99	
CRITICAL VALUE PHONED TO*AC AT 0800 BY GKJ						
RBC	3.35	L	(4.00-5.20)	MILL/C	01/21/99	05:0
HEMOGLOBIN	10.0	L	(11.5-15.5)	G%	01/21/99	05:0
HEMATOCRIT	28.8	L	(35.0-45.0)	%	01/21/99	05:0
MCV	85.9		(77-95)	CMU	01/21/99	05:0
MCH	29.9		(25-33)	MCGM	01/21/99	05:0
MCHC	34.8		(31-37)	%	01/21/99	05:0
RDW	12.8		(11.5-14.5)	%	01/21/99	05:0
WBC DIFF					01/21/99	05:0
WBC MORPH					01/21/99	05:0
WBC DECREASED; NO DIFFERENTIAL PERFORMED					01/21/99	05:0
RBC MORPH	NORMAL				01/21/99	05:0
PLT COUNT	50	L	(140-440)	TH/CMM	01/21/99	05:0

CHEM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
CL	99	L	(101-111)	MMOL/L	01/21/99	05:0
CO2	29		(23-29)	MMOL/L	01/21/99	05:0
K	4.2		(3.5-5.0)	MMOL/L	01/21/99	05:0
NA	138		(136-145)	MMOL/L	01/21/99	05:0
ANION GAP	14		(10-20)		01/21/99	05:0
BUN	10		(6-19)	MG/DL	01/21/99	05:0

=========GROUP CONTINUED=========
11:19 01/21/99 FROM WKH5,REPRTGF1
JAX32018

RESULTS LISTING
2721920102
MR# 00272192 TOD3 T312B PDM

CHEM PROFILE CONT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
GLUCOSE,TM	106		(70-110)	MG/DL	01/21/99	05:0
CREATININE	0.3	L	(0.5-1.1)	MG/DL	01/21/99	05:0

=========END OF REPORT=========
11:19 01/21/99 FROM WKH5,REPRTGF1
JAX32018

RESULTS LISTING
2721920102 MR# 00272192 TODS T312B PDM

BLOOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TI
WBC	0.2	L	(5.0-14.5)	TH/CMM	01/22/99	05
CRITICAL VALUE PHONED TO*CS AT 0705 BY NMB						
RBC	3.05	L	(4.00-5.20)	MILL/C	01/22/99	05
HEMOGLOBIN	9.1	L	(11.5-15.5)	GX	01/22/99	05
HEMATOCRIT	25.6	L	(35.0-45.0)	%	01/22/99	05
MCV	83.9		(77-95)	CMU	01/22/99	05
MCH	29.8		(25-33)	MCGM	01/22/99	05
MCHC	35.5		(31-37)	%	01/22/99	05
RDW	12.5		(11.5-14.5)	%	01/22/99	05
WBC DIFF					01/22/99	05
WBC MORPH					01/22/99	05
WBC DECREASED; NO DIFFERENTIAL PERFORMED						
PLT COUNT	28	L	(140-440)	TH/CMM	01/22/99	05

CHEM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TI
CL	102		(101-111)	MMOL/L	01/22/99	05
CO2	28		(23-29)	MMOL/L	01/22/99	05
K	4.5		(3.5-5.0)	MMOL/L	01/22/99	05
NA	140					
ANION GAP	15		(10-20)		01/22/99	05
BUN	11		(6-19)	MG/DL	01/22/99	05
GLUCOSE,TM	105		(70-110)	MG/DL	01/22/99	05

=GROUP CONTINUED=

08:52 01/22/99 FROM WKJ3,REPRTGF1
32WB3332

RESULTS LISTING
2721920102 MR# 00272192 TODS T312B PDM

CHEM PROFILE CONT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TI
CREATININE	0.4	L	(0.5-1.1)	MG/DL	01/22/99	05

low grade
temp 5.6/24.8
Vanco \ levels ?
Tobra
0.5/4.7

=END OF REPORT=

08:52 01/22/99 FROM WKJ3,REPRTGF1
32WB3332

RESULTS LIST[illegible]
2721920102 TOD3 T312B PDM
MR# 00272192

BLOOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
WBC	0.1	L	(5.0-14.5)	TH/CMM	01/24/99	05:00
CRITICAL VALUE PHONED TO*DB AT 0635 BY NMB						
RBC	2.62	L	(4.00-5.20)	MILL/C	01/24/99	05:00
HEMOGLOBIN	7.9	L	(11.5-15.5)	G%	01/24/99	05:00
HEMATOCRIT	22.4	L	(35.0-45.0)	%	01/24/99	05:00
MCV	85.4		(77-95)	CMU	01/24/99	05:00
MCH	30.3		(25-33)	MCGM	01/24/99	05:00
MCHC	35.4		(31-37)	%	01/24/99	05:00
RDW	12.0		(11.5-14.5)	%	01/24/99	05:00
WBC DIFF					01/24/99	05:00
WBC MORPH					01/24/99	05:00
WBC DECREASED; NO DIFFERENTIAL PERFORMED						
PLT COUNT	60	L	(140-440)	TH/CMM	01/24/99	05:00

CHEM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
CL	104		(101-111)	MMOL/L	01/24/99	05:00
CO2	26		(23-29)	MMOL/L	01/24/99	05:00
K	4.1		(3.5-5.0)	MMOL/L	01/24/99	05:00
NA	140		(136-145)	MMOL/L	01/24/99	05:00
ANION GAP	14		(10-20)		01/24/99	05:00
BUN	13		(6-19)	MG/DL	01/24/99	05:00
GLUCOSE,TM	90		(70-110)	MG/DL	01/24/99	05:00

=GROUP CONTINUED=

1:33 01/24/99 FROM WKH1,REPRTGF1
JAX32564

RESULTS LIST[illegible]
2721920102 TOD3 T312B PDM
MR# 00272192

CHEM PROFILE CONT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
CREATININE	0.4	L	(0.5-1.1)	MG/DL	01/24/99	05:0

=END OF REPORT=

11:33 01/24/99 FROM WKH1,REPRTGF1
WAX32564

2721920094 ORDER/ACC#: 7/001 LAST UPDATE: 01/25/99 12:
-PAGE
SPECIMEN TYPE: COLLECTION DATE/TIME: 01/25/99 11:0

TEST NAME	VALUE	ABN	NORMAL RANGE	UNITS	STS
NEUTROPHIL %	19.1	L	(42-75)	%	F
NEUTRO ABS	0.0	L	(1.4-6.5)	TH/CMM	F
LYMPH %	73.9	H	(21-51)	%	F
LYMPH ABS	0.2	L	(1.2-3.4)	TH/CMM	F
MONONUCL %	4.7		(2-9)	%	F
MONONUCL ABS	0.0			TH/MS	F
EOS %	2.3		(1-4)	%	F
EOS ABS	0.0			TH/MS	F
BASO %	0.0		(0-2)	%	F
BASO ABS	0.0			TH/MS	F

2721920094 ORDER/OCC#: 1/001 LAST UPDATE: 01/25/99 12:2

SPECIMEN TYPE: COLLECTION DATE/TIME: 01/25/99 11:0 -PAGE

TEST NAME	VALUE	ABN	NORMAL RANGE	UNITS	STS
LDH	175		(118-273)	U/L	F

12:21 01/25/99 FROM ;+-,REERPRF6
32WB3523

** STAT ORDER **

RESULTS FOR: THMONC

2721920094 ORDER/OCC#: 3/001 LAST UPDATE: 01/25/99 12:2

SPECIMEN TYPE: COLLECTION DATE/TIME: 01/25/99 11:0 -PAGE

TEST NAME	VALUE	ABN	NORMAL RANGE	UNITS	STS
SGPT	71	H	(0-45)	U/L	F

12:21 01/25/99 FROM ;+-,REERPRF6
32WB3524

** STAT ORDER **

RESULTS FOR: THMONC

2721920094 ORDER/OCC#: 4/001 LAST UPDATE: 01/25/99 12:2

SPECIMEN TYPE: COLLECTION DATE/TIME: 01/25/99 11:0 -PAGE

TEST NAME	VALUE	ABN	NORMAL RANGE	UNITS	STS
URIC ACID	3.5		(3.4-7.0)	MG/DL	F

RESULTS FOR: * * S T A T O R D E R * * THMONC

2721920094 ORDER/OCC#: 5/001 LAST UPDATE: 01/25/99 12:42
-PAGE
SPECIMEN TYPE: COLLECTION DATE/TIME: 01/25/99 11:03

TEST NAME	VALUE	ABN	NORMAL RANGE	UNITS	STS
WBC	0.2 L	L	(5.0-14.5)	TH/CMM	F
CRITICAL VALUE PHONED TO*CV AT 1240 BY BRM					
RBC	2.60	L	(4.00-5.20)	MILL/C	F
HEMOGLOBIN	7.7 L	L	(11.5-15.5)	G%	F
ABNORMAL RESULT PHONED TO*CV AT 1240 BY BRM					
HEMATOCRIT	21.7	L	(35.0-45.0)	%	F
ABNORMAL RESULT PHONED TO*CV AT 1240 BY BRM					
MCV	83.5		(77-95)	CMU	F
MCH	29.8		(25-33)	MCGM	F
MCHC	35.7		(31-37)	%	F
RDW	11.7		(11.5-14.5)	%	F
PLT COUNT	40 L	L	(140-440)	TH/CMM	F

12:42 01/25/99 FROM ;+-,REERPRF6
32WB3541

2721920094 ORDER/OCC#: 2/001 LAST UPDATE: 01/25/99 12:2
-PAGE
SPECIMEN TYPE: COLLECTION DATE/TIME: 01/25/99 11:0

TEST NAME	VALUE	ABN	NORMAL RANGE	UNITS	STS
CL	101 L		(101-111)	MMOL/L	F
CO2	27 L		(23-29)	MMOL/L	F
K	4.0 L		(3.5-5.0)	MMOL/L	F
NA	137 L		(136-145)	MMOL/L	F
ANION GAP	13 L		(10-20)		F
BUN	16		(6-19)	MG/DL	F
GLUCOSE,TM	103 L		(70-110)	MG/DL	F
PROTEIN,TOT.	7.2 L		(6.0-8.5)	GM/DL	F
ALBUMIN	4.4 L		(3.9-4.8)	GM/DL	F
CA	9.8 L		(8.5-10.5)	MG/DL	F
CREATININE	0.3 L	L	(0.5-1.1)	MG/DL	F
BILI,TOTAL	0.7 L		(0.1-1.5)	MG/DL	F
ALK PHOS	171 L		(117-390)	U/L	F
SGOT	47 L	H	(0-37)	U/L	F

RESULTS FOR: * * S T A T O R D E R * * THMONC

2721920094 ORDER/OCC#: 2/001 LAST UPDATE: 01/25/99 12:2
-PAGE
SPECIMEN TYPE: COLLECTION DATE/TIME: 01/25/99 11:0

TEST NAME	VALUE	ABN	NORMAL RANGE	UNITS	STS
CL	101		(101-111)	MMOL/L	F
CO2	27		(23-29)	MMOL/L	F
K	4.0		(3.5-5.0)	MMOL/L	F
NA	137		(136-145)	MMOL/L	F
ANION GAP	13		(10-20)		F
BUN	16		(6-19)	MG/DL	F
GLUCOSE,TM	103		(70-110)	MG/DL	F
PROTEIN,TOT.	7.2		(6.0-8.5)	GM/DL	F
ALBUMIN	4.4		(3.9-4.8)	GM/DL	F
CA	9.8		(8.5-10.5)	MG/DL	F
CREATININE	0.3	L	(0.5-1.1)	MG/DL	F
BILI,TOTAL	0.7		(0.1-1.5)	MG/DL	F
ALK PHOS	171		(117-390)	U/L	F
SGOT	47	H	(0-37)	U/L	F

12:21 01/25/99 FROM ;+-,REERPRF6
32WB3522

* * STAT ORDER * *

RESULTS FOR: THMONC

2721920094 ORDER/OCC#: 2/001 LAST UPDATE: 02/08/99 10 -PAGE

SPECIMEN TYPE: COLLECTION DATE/TIME: 02/08/99 10:0

TEST NAME	VALUE	ABN	NORMAL RANGE	UNITS	STS
CL	105		(101-111)	MMOL/L	F
CO2	25		(23-29)	MMOL/L	F
K	4.3		(3.5-5.0)	MMOL/L	F
NA	138		(136-145)	MMOL/L	F
ANION GAP	12		(10-20)		F
BUN	11		(6-19)	MG/DL	F
GLUCOSE,TM	99		(70-110)	MG/DL	F
PROTEIN,TOT.	6.3		(6.0-8.5)	GM/DL	F
ALBUMIN	4.2		(3.9-4.8)	GM/DL	F
CA	9.5		(8.5-10.5)	MG/DL	F
CREATININE	0.4	L	(0.5-1.1)	MG/DL	F
BILI,TOTAL	0.5		(0.1-1.5)	MG/DL	F
ALK PHOS	257		(117-390)	U/L	F
SGOT	25		(0-37)	U/L	F

10:53 02/08/99 FROM ;+-,REERPRF6
32WB5550

* * STAT ORDER * *

RESULTS FOR: THMONC

2721920094 ORDER/OCC#: 1/001 LAST UPDATE: 02/08/99 10:5 -PAGE

SPECIMEN TYPE: COLLECTION DATE/TIME: 02/08/99 10:0

TEST NAME	VALUE	ABN	NORMAL RANGE	UNITS	STS
LDH	190		(118-273)	U/L	F

10:53 02/08/99 FROM ;+-,REERPRF6
32WB5551

1

Tuesday

* * S T A T O R D E R * *

LTS FOR: THMONC

721920094 ORDER/OCC#: 5/001 LAST UPDATE: 02/09/99 10:35

CIMEN TYPE: COLLECTION DATE/TIME: 02/09/99 09:31 PAGE 1

EST NAME	VALUE	ABN	NORMAL RANGE	UNITS	STS
	1.1	L	(5.0-14.5)	TH/CMM	F
	[illegible]	L	(4.00-5.20)	MILL/C	F
OGLOBIN	7.8	L	(11.5-15.5)	G%	F
		ABNORMAL RESULT PHONED TO TE AT 1036			
		ABNORMAL RESULT PHONED TO TE AT 1036 BY VR			
ATOCRIT	22.7	L	(35.0-45.0)	%	F
NORMAL RESULT PHONED TO*TE AT 1036 BY VR					
	80.6		(77-95)	CMU	F
	27.6		(25-33)	MCGM	F
C	34.2		(31-37)	%	F
	12.2		(11.5-14.5)	%	F
COUNT	36	L	(140-440)	TH/CMM	F

5 02/09/99 FROM ;+-,REERPRF6
5986

* * S T A T O R D E R * *

LTS FOR: THMONC

721920094 ORDER/OCC#: 5/001 LAST UPDATE: 02/09/99 10:36

CIMEN TYPE: COLLECTION DATE/TIME: 02/09/99 09:31 PAGE 1

EST NAME	VALUE	ABN	NORMAL RANGE	UNITS	STS
DIFF					F
TROPHIL %	43.3		(42-75)	%	F
TRO ABS	0.5	L	(1.4-6.5)	TH/CMM	F
PH %	25.3		(21-51)	%	F
PH ABS	0.3	L	(1.2-3.4)	TH/CMM	F
ONUCL %	29.6	H	(2-9)	%	F
ONUCL ABS	0.3			TH/M3	F
%	0.8	L	(1-4)	%	F
ABS	0.0			TH/M3	F
O %	1.0		(0-2)	%	F
O ABS	0.0			TH/M3	F

* Stop K completely
✓ Thurs. -

6 02/09/99 FROM ;+-,REERPRF6
5987

* * S T A T O R D E R * *

ULTS FOR: [illegible] THMONC

2721920094 ORDER/OCC#: 2/001 LAST UPDATE: 02/09/99 10:34
ECIMEN TYPE: COLLECTION DATE/TIME: 02/09/99 09:31 -PAGE 1

TEST NAME	VALUE	ABN	NORMAL RANGE	UNITS	STS
	106		(101-111)	MMOL/L	F
2	28		(23-29)	MMOL/L	F
	4.4		(3.5-5.0)	MMOL/L	F
	138		(136-145)	MMOL/L	F
ION GAP	8	L	(10-20)		F
N	11		(6-19)	MG/DL	F
UCOSE,TM	81		(70-110)	MG/DL	F
OTEIN,TOT.	6.1		(6.0-8.5)	GM/DL	F
BUMIN	3.9		(3.9-4.8)	GM/DL	F
	9.2		(8.5-10.5)	MG/DL	F
EATININE	0.4	L	(0.5-1.1)	MG/DL	F
LI,TOTAL	0.5		(0.1-1.5)	MG/DL	F
K PHOS	246		(117-390)	U/L	F
OT	24		(0-37)	U/L	F

34 02/09/99 FROM ;+-,REERPRF6
B5982

* * S T A T O R D E R * *

ULTS FOR: THMONC

2721920094 ORDER/OCC#: 1/001 LAST UPDATE: 02/09/99 10:34
ECIMEN TYPE: COLLECTION DATE/TIME: 02/09/99 09:31 -PAGE 1

TEST NAME	VALUE	ABN	NORMAL RANGE	UNITS	STS
H	195		(118-273)	U/L	F

34 02/09/99 FROM ;+-,REERPRF6

ESULTS FOR: * * S T A T O R D E R * *

THMONC

2721920094 ORDER/OCC#: 3/001 LAST UPDATE: 02/09/99 10:35

SPECIMEN TYPE: COLLECTION DATE/TIME: 02/09/99 09:31 -PAGE 1

TEST NAME	VALUE	ABN	NORMAL RANGE	UNITS	STS
SGPT	32		(0-45)	U/L	F

):35 02/09/99 FROM ;+-,REERPRF6
2WB5984

ESULTS FOR: * * S T A T O R D E R * *

THMONC

2721920094 ORDER/OCC#: 4/001 LAST UPDATE: 02/09/99 10:35

SPECIMEN TYPE: COLLECTION DATE/TIME: 02/09/99 09:31 -PAGE 1

TEST NAME	VALUE	ABN	NORMAL RANGE	UNITS	STS
URIC ACID	4.2		(3.4-7.0)	MG/DL	F

):35 02/09/99 FROM ;+-,REERPRF6

BLOOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	T
WBC	2.0	L	(5.0-14.5)	TH/CMM	03/03/99	05
RBC	3.20	L	(4.00-5.20)	MILL/C	03/03/99	05
HEMOGLOBIN	10.3	L	(11.5-15.5)	G%	03/03/99	05
HEMATOCRIT	30.3	L	(35.0-45.0)	%	03/03/99	05
MCV	94.5		(77-95)	CMU	03/03/99	05
MCH	32.2		(25-33)	MCGM	03/03/99	05
MCHC	34.1		(31-37)	%	03/03/99	05
RDW	25.6	H	(11.5-14.5)	%	03/03/99	05
WBC DIFF					03/03/99	05
POLY %	28	L	(50-70)	%	03/03/99	05
STAB %	10	H	(2-6)	%	03/03/99	05
EOSINOPHIL	9	H	(0-4)	%	03/03/99	05
BASOPHIL %	1		(0-2)	%	03/03/99	05
LYMPHOCYTE	32		(20-44)	%	03/03/99	05
MONOCYTES %	20	H	(2-9)	%	03/03/99	05
RBC MORPH					03/03/99	05
SEV*ANISO*SLT*POLY*SLT*TEAR DROP*SLT*ELLIPT*SLT*SPHERO					03/03/99	05
PLT COUNT	210		(140-440)	TH/CMM	03/03/99	05

CHEM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE	T
CL	105		(101-111)	MMOL/L	03/03/99	05
CO2	25		(23-29)	MMOL/L	03/03/99	05

==============================GROUP CONTINUED==============================

08:45 03/03/99 FROM WAS1,REPRTGF1
32WB9549

RESULTS LISTING
2721920151 TOD2 T207B HEM
MR# 00272192

CHEM PROFILE CONT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	T
K	3.6		(3.5-5.0)	MMOL/L	03/03/99	05
NA	141		(136-145)	MMOL/L	03/03/99	05
ANION GAP	15		(10-20)		03/03/99	05
BUN	17		(6-19)	MG/DL	03/03/99	05
GLUCOSE,TM	83		(70-110)	MG/DL	03/03/99	05
CREATININE	0.4	L	(0.5-1.1)	MG/DL	03/03/99	05

==============================END OF REPORT==============================

08:45 03/03/99 FROM WAS1,REPRTGF1
32WB9549

BLOOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	T
WBC	0.2	L	(5.0-14.5)	TH/CMM	03/23/99	0
CRITICAL VALUE PHONED TO*SD AT 0840 BY NMB						
RBC	3.00	L	(4.00-5.20)	MILL/C	03/23/99	0
HEMOGLOBIN	9.1	L	(11.5-15.5)	G%	03/23/99	0
HEMATOCRIT	25.4	L	(35.0-45.0)	%	03/23/99	0
MCV	84.7		(77-95)	CMU	03/23/99	0
MCH	30.3		(25-33)	MCGM	03/23/99	0
MCHC	35.8		(31-37)	%	03/23/99	0
RDW	16.5	H	(11.5-14.5)	%	03/23/99	0

Plt 21 unverified

CHEM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE	T
CL	103		(101-111)	MMOL/L	03/23/99	0
CO2	26		(23-29)	MMOL/L	03/23/99	0
K	4.2		(3.5-5.0)	MMOL/L	03/23/99	0
NA	138		(136-145)	MMOL/L	03/23/99	0
ANION GAP	13		(10-20)		03/23/99	0
BUN	8		(6-19)	MG/DL	03/23/99	0
GLUCOSE,TM	78		(70-110)	MG/DL	03/23/99	0
CREATININE	0.4	L	(0.5-1.1)	MG/DL	03/23/99	0
PHOSPHOROUS	4.8		(4.5-6.5)	MG/DL	03/23/99	0
CA	9.2		(8.5-10.5)	MG/DL	03/23/99	0

==============================END OF REPORT==============================

08:48 03/23/99 FROM WKJ3,REPRTGF1

BLOOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
WBC	3.7	L	(5.0-14.5)	TH/CMM	03/04/99	05:0
RBC	3.41	L	(4.00-5.20)	MILL/C	03/04/99	05:0
HEMOGLOBIN	11.0	L	(11.5-15.5)	G%	03/04/99	05:0
HEMATOCRIT	32.0	L	(35.0-45.0)	%	03/04/99	05:0
MCV	94.0		(77-95)	CMU	03/04/99	05:0
MCH	32.3		(25-33)	MCGM	03/04/99	05:0
MCHC	34.4		(31-37)	%	03/04/99	05:0
RDW	25.0	H	(11.5-14.5)	%	03/04/99	05:0
WBC DIFF					03/04/99	05:0
POLY %	75	H	(50-70)	%	03/04/99	05:0
STAB %	13	H	(2-6)	%	03/04/99	05:0
EOSINOPHIL	2		(0-4)	%	03/04/99	05:0
LYMPHOCYTE	3	L	(20-44)	%	03/04/99	05:0
MONOCYTES %	7		(2-9)	%	03/04/99	05:0
RBC MORPH					03/04/99	05:0
SEV*ANISO*SLT*HYPO*MOD*POLY*SLT*SPHERO*SLT*TEAR DROP						
PLT COUNT	211		(140-440)	TH/CMM	03/04/99	05:0

CHEM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
CL	104		(101-111)	MMOL/L	03/04/99	05:0
CO2	23		(23-29)	MMOL/L	03/04/99	05:0
K	3.8		(3.5-5.0)	MMOL/L	03/04/99	05:0

=========GROUP CONTINUED=========

9:25 03/04/99 FROM WKH5,REPRTGF1
AX39842

RESULTS LISTING
2721920151 TOD2 T207B HEM
MR# 0027z192

CHEM PROFILE CONT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TIME
NA	139		(136-145)	MMOL/L	03/04/99	05:0
ANION GAP	16		(10-20)		03/04/99	05:0
BUN	10		(6-19)	MG/DL	03/04/99	05:0
GLUCOSE,TM	96		(70-110)	MG/DL	03/04/99	05:0
CREATININE	0.3	L	(0.5-1.1)	MG/DL	03/04/99	05:0

emesis
DS .45
10 KCl

88
37
616
2640
ANC 3256

=========END OF REPORT=========

9:25 03/04/99 FROM WKH5,REPRTGF1
AX39842

BLOOD COUNT	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TI
WBC	3.6	L	(5.0-14.5)	TH/CMM	03/05/99	05
RBC	3.02	L	(4.00-5.20)	MILL/C	03/05/99	05
HEMOGLOBIN	9.7	L	(11.5-15.5)	G%	03/05/99	05
HEMATOCRIT	28.6	L	(35.0-45.0)	%	03/05/99	05
MCV	93.5		(77-95)	CMU	03/05/99	05
MCH	32.3		(25-33)	MCGM	03/05/99	05
MCHC	34.5		(31-37)	%	03/05/99	05
RDW	23.9	H	(11.5-14.5)	%	03/05/99	05

103
99

CHEM PROFILE	VALUE	ABN	NORMAL RANGE	UNIT	DATE	TI
CL	104		(101-111)	MMOL/L	03/05/99	05
CO2	25		(23-29)	MMOL/L	03/05/99	05
K	3.7		(3.5-5.0)	MMOL/L	03/05/99	05
NA	139		(136-145)	MMOL/L	05/05/99	05
ANION GAP	14		(10-20)		03/05/99	05
BUN	7		(6-19)	MG/DL	03/05/99	05
GLUCOSE,TM	101		(70-110)	MG/DL	03/05/99	05
CREATININE	0.3	L	(0.5-1.1)	MG/DL	03/05/99	05

=========END OF REPORT=========

08:34 03/05/99 FROM WKJ3,REPRTGF1

ISULTS FOR: ** STAT ORDER **

THMONC

278192021 9 ORDER/OCC#: 1/001 LAST UPDATE: 04/15/99 10:03

SPECIMEN TYPE: PAGE 1

COLLECTION DATE/TIME: 04/15/99 08:41

TEST NAME	VALUE	ABN	NORMAL RANGE	UNITS	STS
'BC	0.5	L	(5.0-14.5)	TH/CMM	F
CRITICAL VALUE PHONED TO*TE AT 1000 BY NMB					
BC	3.82	L	(4.00-5.20)	MILL/C	F
EMOGLOBIN	11.0	L	(11.5-15.5)	G%	F
EMATOCRIT	31.0	L	(35.0-45.0)	%	F
CV	81.0		(77-95)	CMU	F
CH	28.8		(25-33)	MCGM	F
CHC	35.6		(31-37)	%	F
DW	13.1		(11.5-14.5)	%	F
LT COUNT	77	L	(140-440)	TH/CMM	F

:05 04/15/99 FROM ;**;REERPRPA

166376

ULTS FOR: ** STAT ORDER **

THMONC

278192021 9 ORDER/OCC#: 1/001 LAST UPDATE: 04/15/99 10:05

ECIMEN TYPE: PAGE

COLLECTION DATE/TIME: 04/15/99 08:41

TEST NAME	VALUE	ABN	NORMAL RANGE	UNITS	STS
C DIFF					
UTROPHIL %	42.0		(42-75)	%	F
UTRO ABS	0.2	L	(1.4-6.5)	TH/CMM	F
MPH %	16.6	L	(20-51)	%	F
MPH ABS	0.1	L	(1.2-3.4)	TH/CMM	F
MONUCL %	35.4	H	(2-9)	%	F
MONUCL ABS	0.2			TH/MS	F
S %	2.0		(1-4)	%	F
S ABS	0.0			TH/MS	F
SO %	4.0	H	(0-2)	%	F
SO ABS	0.0			TH/MS	F

5 04/15/99 FROM ;**;REERPRPA

6377

ESULTS LISTING
2721920268 MEM

MR# 00272192 THMONC

	07/13/99	06/29/99	06/15/99
BLOOD COUNT	08:54	12:15	08:25
WBC	4.1*	4.5*	3.4*
RBC	4.34	4.43	4.13
HEMOGLOBIN	14.0	14.6	14.0
HEMATOCRIT	40.3	41.5	39.4
MCV	92.8	93.7	95.4*
MCH	32.4	33.0	33.9*
MCHC	34.9	35.2	35.5
RDW	11.3*	11.9	13.9
DIFFERENTIAL			
WBC DIFF			
NEUTROPHIL %	51.3	55.6	42.6
NEUTRO ABS	2.1	2.4	1.5
LYMPH %	21.9	18.6*	22.2
LYMPH ABS	0.9*	0.8*	0.8*
MONONUCL %	10.4*	11.4*	12.9*
MONONUCL ABS	0.4	0.5	0.4
EOS %	15.9*	14.3*	21.0*
EOS ABS	0.6	0.6	0.7
BASO %	0.0	0.1	0.0
BASO ABS	0.0	0.1	0.0
POLY %			45*

GROUP CONTINUED

40 07/13/99 FROM MAS1,REPRTGFL
80272

ESULTS LISTING
2721920268 MEM

MR# 00272192 THMONC

	07/13/99	06/29/99	06/15/99
BLOOD COUNT CON	08:54	12:15	08:25
STAB %			3
EOSINOPHIL %			15*
BASOPHIL %			1
LYMPHOCYTE %			15*
ATYP/REACT %			3
MONOCYTES %			10*
RBC MORPH		NORMAL	SEE TEXT
PLT COUNT	221	279	248

	07/13/99	06/15/99
HEMATOLOGY	07:15	07:15
BONE M.SPEC:	SEE TEXT	SEE TEXT

	06/15/99
MISC. TESTS	13:00
TEST NAME	SEE TEXT
RESULTS CONT	SEE TEXT

END OF REPORT

40 07/13/99 FROM MAS1,REPRTGFL
80272

APPENDIX 3

Environmental Data: Map of Berlin Lake, EPA documentation of spills into Berlin Lake Reservoir

MAP OF BERLIN LAKE

BERLIN LAKE

1 1/2 0 1

SCALE IN MILES

US Army Corps
of Engineers
Pittsburgh District

Revised: 1993

RECREATION AREAS

CORPS OF ENGINEERS

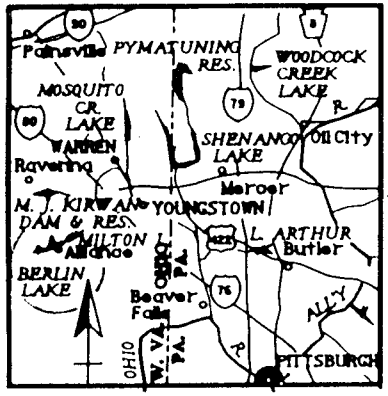
- Dam Picnic Area
- Mill Creek Rec Area
- Resource Managers Office
- German Church Campground

COMMERCIAL

(5) Dutch Harbor Marina and Concessionair
(6) Lee's Bait Shop

OTHER AGENCY

(7) Deer Creek Rec. Area
(8) Willow Creek Rec. Area
(9) Bonner Road Boat Launching Ramp



VICINITY MAP

25 0 50

SCALE IN MILES

N

To Cleveland

Yale Rd.

PORTAGE CO.
OHIO

To Ohio Rte. 18

Alliance Rd.

Wayland Rd.

Notman Rd.

McClintocksburg Rd.

Jones Rd.

MILTON LAKE

Ellsworth Rd.

Woodward Dr.

E. River Rd.

MAHONING R.

Bedell Rd.

Mock Rd.

MAHONING CO.
OHIO

Helser Rd.

Mottown Rd.

DAM

Mahoning Rd.

Bonner Rd.

Turkeystrout Cr.

Deerfield

To Youngstown

225

224

14

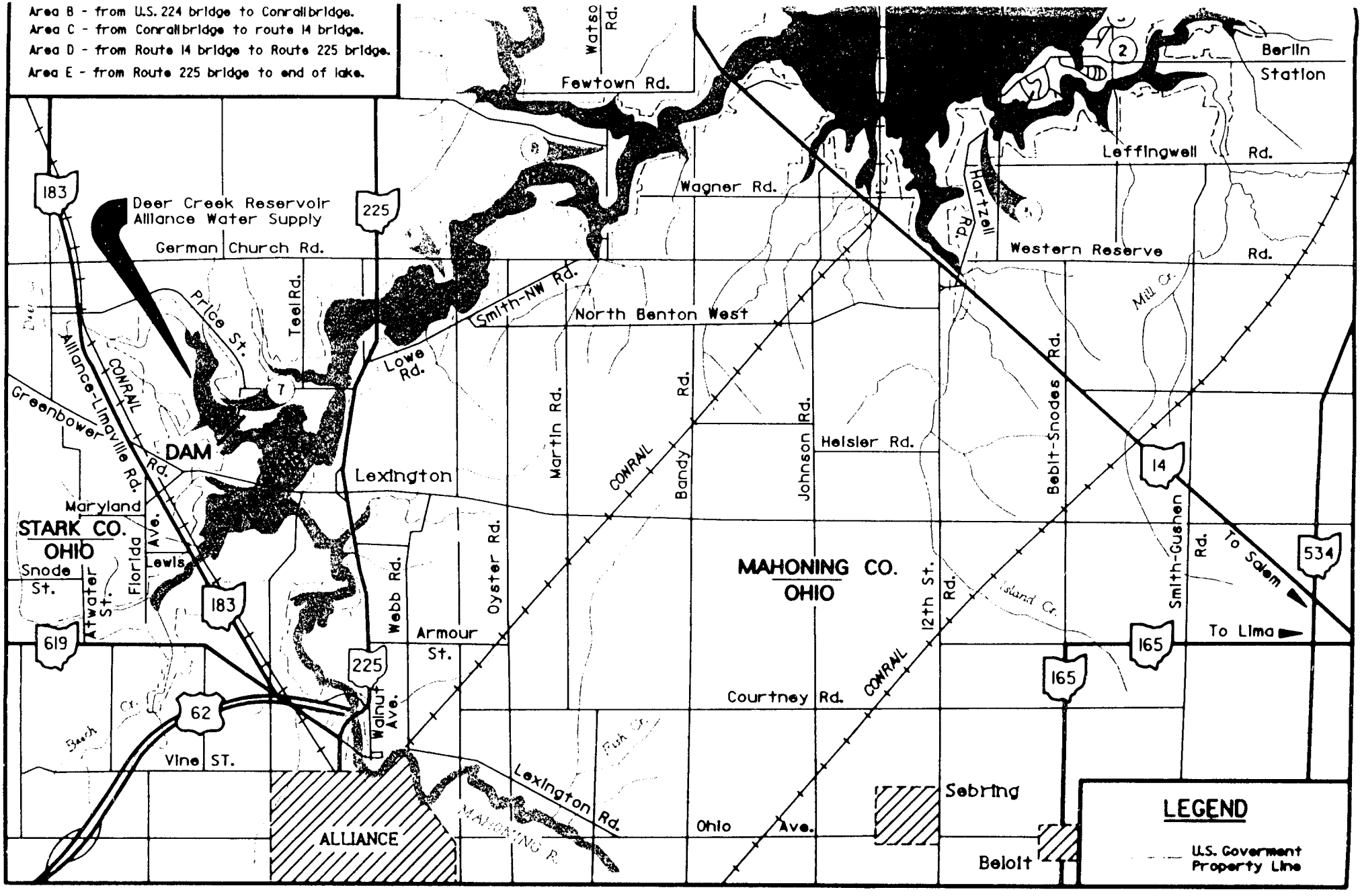
1

9

6

NOTE

No motors permitted within 2000 feet of the dam.
No boats permitted within 500 feet of the intake tower and crest gate, it is unlawful to cause a wake within 150 feet of existing waterline or bridge.


Area B - from U.S. 224 bridge to Conrail bridge.
Area C - from Conrail bridge to route 14 bridge.
Area D - from Route 14 bridge to Route 225 bridge.
Area E - from Route 225 bridge to end of lake.
Watso Rd.
Fewtown Rd.
Berlin
Station
Leffingwell Rd.
Wagner Rd.
Hartzell Rd.
Western Reserve Rd.
183
225
Deer Creek Reservoir
Alliance Water Supply
German Church Rd.
Price St.
Teel Rd.
Smith-NW Rd.
North Benton West
Mill Cr.
Lowe Rd.
Alliance-Limaville Rd.
CONRAIL
Greenbower Rd.
DAM
Lexington
Martin Rd.
Bandy Rd.
Johnson Rd.
Helsler Rd.
Bebit-Shodes Rd.
14
Maryland
STARK CO.
OHIO
Snode St.
Atwater St.
Florida Ave.
Lewis
Webb Rd.
Oyster Rd.
MAHONING CO.
OHIO
Armour St.
12th St.
Smith-Gusehen Rd.
To Salem
534
To Lima
619
183
225
165
165
62
Walnut Ave.
Courtney Rd.
Island Cr.
Fish Cr.
Vine ST.
Lexington Rd.
Sebring
ALLIANCE
Ohio Ave.
Beloit
MAHONING R.
LEGEND
U.S. Goverment Property Line

EPA DOCUMENTATION
OF SPILLS INTO BERLIN LAKE RESERVOIR

ENTITY	COUNTY	TWP_CITY	WATERWAY	MATERIAL_1	AMOUNT_1	UNITS_1	RECOVER_1
A Y S MEDICAL EQUIPMENT IN	MAHONING	YOUNGSTOWN	STORM SEWER	RED DYE	0	UNK	0
A-1 AUTO BODY	PORTAGE	FREEDOM TWP	UNKNOWN CREEK	OIL	0	UNK	0
AEROLIKE EXTRUSION CO	MAHONING	BOARDMAN TWP	MAHONING RIVER	SODIUM HYDROXIDE SOLU	50	GAL	0
AEROLITE EXTRUSION CO	MAHONING	BOARDMAN	UNKNOWN	CAUSTIC SODA	0	UNK	0
ALL AMERICAN TRACK CO	MAHONING	BOARDMAN	MILL CREEK	SUBSTANCE 4170	400	GAL	0
ALL AMERICAN TRACK CO	MAHONING	BOARDMAN	SAWMILL RUN CREEK	LATEX PAINT	300	GAL	0
ALLIANCE BOARD OF ED.	STARK	ALLIANCE	MAHONING RIVER	STYRENE BUTADIENE POLY	5	GAL	0
ALLIANCE STP	STARK	ALLIANCE	MAHONING RIVER TRIB	OIL	20	GAL	0
ALLIANCE STP	STARK	ALLIANCE	BEECH CREEK TRIB	DIESEL FUEL	120	GAL	120
ALLIANCE TUBULAR	STARK	ALLIANCE	MAHONING RIVER	MILKY WHITE STUFF	0	UNK	0
ALLIANCE WATER DEPT	STARK	ALLIANCE	DEER CREEK	HYDRAULIC OIL	100	GAL	0
ALSID OIL & GAS CO	MAHONING	SMITH TWP	BELOIT DITCH	CRUDE OIL	2	GAL	0
ALUMINUM COLOR INDUSTRIE	MAHONING	LOWELLVILLE	MAHONING RIVER TRIB	WASTE ACID	0	UNK	0
AMERICAN PAPER PRODUCTS	MAHONING	YOUNGSTOWN	SEWER	COUSTIC SODA	0	UNK	0
AMERISOURCE	MAHONING	YOUNGSTOWN	UNKNOWN	TRANSFORMER OIL	0	UNK	0
AMOCO OIL CO	PORTAGE	GANETESVILLE	SILVER CREEK	MOTOR OIL	0	UNK	0
ANDEL RESIDENCE	PORTAGE	GARRETTSVILLE	MAHONING RIVER TRIB	SEPTIC TANK SOLIDS	0	UNK	0
ARCO PIPELINE	MAHONING	N.JACKSON	UNKNOWN	FUEL OIL #2	1252	GAL	0
ASHLAND BRANDED MARKETI	STARK	ALLIANCE	STORM SEWER	KEROSINE	1	GAL	0
ASHLAND OIL CO.	TRUMBULL	WARREN	STORM SEWER	#2 FUEL OIL	5	GAL	0
AUTUMN IND	TRUMBULL	WARREN	SWAMP	HAZARDOUS WASTE RESID	0	UNK	0
AVILA CONTRACTING	TRUMBULL	LIBERTY TWP	UNKNOWN CREEK	POND SLUDGE	0	UNK	0
B F I	PORTAGE	ATWATER	BERLIN RESERVOIR	GARBAGE	0	UNK	0
B F I	PORTAGE	ATWATER	BERLIN RESERVOIR	GARBAGE	0	UNK	0
B F I	PORTAGE	ATWATER	BERLIN RESERVOIR	LEACHATE	0	UNK	0
B F I	PORTAGE	ATWATER	MILL CREEK	ODORS	0	UNK	0
B F I	PORTAGE	ATTWATER	WILLOW CREEK	ODOR	0	UNK	0
B F I	STARK	LEXINGTON TWP	UNKNOWN	HYDRAULIC OIL	7550	GAL	0
B F I	MAHONING	BERLIN CENTER	UNKNOWN	HYDRAULIC OIL	40	GAL	0
B F I	PORTAGE	ATWATER	BERLEN RESERVIOR	SULFUR DIOXIDE	0	UNK	0
B P OIL CO / PIPELINE DIV	TRUMBULL	LORDSTOWN TW	STORM SEWER	DIESEL FUEL	8400	GAL	0
B-RIGHT TRUCKING CO	MAHONING	YOUNGSTOWN	STORM SEWER	DIESEL FUEL	50	GAL	50
BABCOCK LUMBER	TRUMBULL	HUBBARD	UNKNOWN	DIESEL FUEL	0	UNK	0
BABCOCKS & WILCOX / TUBUL	STARK	ALLIANCE	RYANS RUN	WASTE SULFURIC ACID	100	GAL	0
BAGETTA TOWNSHIP GARAGE	TRUMBULL	CORTLAND	GROUNDWATER/MOSQUITO RE	SALT	0	UNK	0
BAZETTA TWP	TRUMBULL	BAZETTA TWP	POND	SEPTIC WASTE	0	UNK	0
BEAZER EAST INC	MAHONING	YOUNGSTOWN	CRAB CREEK	TOC 73.5	0	UNK	0
BEAZER EAST INC	MAHONING	YOUNGSTOWN	CRAB CREEK	WASTE WATER	0	UNK	0
BEAZER EAST INC	MAHONING	YOUNGSTOWN	CRAB CREEK	WASTE WATER	0	UNK	0
BEAZER EAST INC	MAHONING	YOUNGSTOWN	GLADE CREEK	WASTE WATER	0	UNK	0
BEAZER EAST INC	MAHONING	YOUNGSTOWN	CRAB CREEK	WASTE WATER	0	UNK	0
BELDON & BLAKE	MAHONING	SMITH TWP	UNKNOWN	CRUDE OIL	0	UNK	0
BELOIT STP	MAHONING	BELOIT	MAHONING RIVER	WASTE WATER	75000	GAL	0
BELOIT STP	MAHONING	BELOIT	BERLIN LAKE TRIB	WASTE WATER	0	UNK	0
BELOIT STP	MAHONING	BELOIT	MAHONING RIVER	WASTE WATER	0	UNK	0
BELOIT STP	MAHONING	BELOIT	MAHONING RIVER	WASTE WATER	0	UNK	0
BELOIT STP	MAHONING	BELOIT	MAHONING RIVER	SEWAGE	0	UNK	0
BELOIT STP	MAHONING	BELOIT	MAHONING RIVER	WASTE WATER	0	UNK	0
BELOIT STP	MAHONING	BELOIT	MAHONING RIVER	SEWAGE	0	UNK	0
BELOIT STP	MAHONING	BELOIT	MAHONING RIVER	WASTE WATER	0	UNK	0
BELOIT STP	MAHONING	BELOIT	MAHONING RIVER	SEWAGE	0	UNK	0
BELOIT STP	MAHONING	BELOIT	MAHONING RIVER	WASTE WATER	0	UNK	0
BELOIT STP	MAHONING	BELOIT	MAHONING RIVER	WASTE WATER	0	UNK	0
BELOIT STP	MAHONING	BELOIT	MAHONING RIVER	WASTE WATER	0	UNK	0
BELOIT STP	MAHONING	BELOIT	MAHONING RIVER	WASTE WATER	0	UNK	0

YR	ENTITY	COUNTY	TWP_CITY	WATERWAY	MATERIAL_1	AMOUNT_1	UNITS_1	RECOVER_1
92	DAYCO / DIETRICH IND	TRUMBULL	WARREN	STORM SEWER	WASTE CHEMICALS	50	GAL	0
90	DAYTON POWER & LIGHT	TRUMBULL	NILES	WETLAND	CRUDE OIL	0	UNK	0
89	DEFT INC	STARK	ALLIANCE	UNKNOWN	STAIN	0	UNK	0
93	DENMAN TIRE & RUBBER	TRUMBULL	BRACEVILLE TW	MAHONING RIVER	DIESEL FUEL	0	UNK	0
89	DENMAN TIRE & RUBBER	TRUMBULL	LEVITSBURG	MAHONING RIVER	CARBON BLACK	0	UNK	0
89	DIAMOND HEAD EXTENDED C	MAHONING	NORTH LIMA	UNKNOWN	SEWAGE	0	UNK	0
91	DON FOSTER & SON CARPET	STARK	ALLIANCE	STORM DRAIN	GREY GREENISH STUFF	0	UNK	0
93	DON FOSTOR & SON CARPET	STARK	ALLIANCE	UNKNOWN CREEK	CLEANING CHEMICALS	0	UNK	0
92	DORFMAN PRODUCTION	MAHONING	DAMASCUS	UNKNOWN	CRUDE OIL	4800	GAL	4000
93	DOUG'S TRUCK & TRAILER	MAHONING	YOUNGSTOWN	UNKNOWN CREEK	OIL	0	UNK	0
91	DOWELL SCHLUMBERGER INC	MAHONING	AUSTINTOWN	SEWER	ACIDS	0	UNK	0
91	DUFF'S CARPET CLEANING	MAHONING	AUSTINTOWN	UNKNOWN	WASTE WATER	0	UNK	0
91	EAGLE CHEVY OLDS	TRUMBULL	HUBBARD	UNKNOWN	ANTIFREEZE	0	UNK	0
93	EARL COREY CO	COLUMBIAN	COLUMBIANA	E BR MILL CREEK	OIL	50	GAL	40
93	EARL COREY CO	COLUMBIAN	COLUMBIANA	UNKNOWN CREEK	MOTOR OIL	0	UNK	0
89	EAST OHIO GAS	MAHONING	DEERFIELD	POND	CRUDE OIL	100	GAL	0
90	EAST OHIO GAS	MAHONING	BERLIN CENTER	BERLIN RESERVOIR	#2 FUEL OIL	0	UNK	0
92	EAST OHIO GAS	MAHONING	AUSTINTOWN TW	MEANDER CK RESV.	NATURAL GAS CONDENSAT	1	GAL	0
92	EAST OHIO GAS	MAHONING	YOUNGSTOWN	STORM SEWER	GASOLINE	4	GAL	0
92	EASTERN EVERFLO	MAHONING	ELLSWORTH TW	MEANDER CREEK	NATURAL GAS	0	UNK	0
91	EASTERN PETROLEUM	TRUMBULL	WEATHERSFIELD	UNKNOWN	CRUDE OIL	0	UNK	0
89	EASTERN PETROLEUM	MAHONING	TRUMBULL CO	UNKNOWN	UNK	0	UNK	0
90	EMRO MARKETING / GAS TOW	MAHONING	NORTH LIMA	UNKNOWN	DIESEL FUEL	25	GAL	0
91	EMRO MARKETING / SPEEDW	MAHONING	AUSTINTOWN	SULFUR RUN TRIB	DIESEL FUEL	0	UNK	0
91	EVERFLOW EASTERN	MAHONING	ALLIANCE	MAHONING RIVER TRIB	DIESEL FUEL	50	GAL	25
92	FAUL & SONS TOOL & DIE CO	TRUMBULL	NILES	UNKNOWN	WASTE OIL	0	UNK	0
92	FFE TRANSPORTATION SERVI	TRUMBULL	WEATHERSFIELD	MAHONING RIVER	DIESEL FUEL	125	GAL	25
89	FISHBURN WELL SERVICE	MAHONING	MINERAL RIDGE	MEANDER CREEK	RUSTY RED MATERIAL	0	UNK	0
89	FITNESS CENTER	TRUMBULL	BAZETTA	STORM SEWER	SWIMMING POOL CHEMICA	0	UNK	0
91	FORMER OPEN PIT MINING/ NA	MAHONING	GOSHEN TWP	MEANDER CREEK TRIB	IRON OXIDE	0	UNK	0
91	FORT INDUSTRIES	MAHONING	YOUNGSTOWN	UNNAMED CREEK	ASBESTOS	0	UNK	0
89	FRANK MARTUCCIO ENTERPRI	MAHONING	MILTON TWP	LAKE MILTON	DIESEL FUEL	200	GAL	100
93	GASTOWN GAS STATION	MAHONING	NEW MIDDLETON	STORM SEWER	GASOLINE	0	UNK	0
90	GENERAL AGGREGATES	TRUMBULL	KINGSMAN	OLD ROCK QUARRY	UNK	0	UNK	0
90	GENERAL ELECTRIC	TRUMBULL	NILES	MOSQUITO CREEK	LUBRICATING OIL	6	GAL	0
92	GENERAL ELECTRIC	TRUMBULL	NILES	MOSQUITO CREEK	HYDRAULIC OIL	5	GAL	0
93	GENERAL ELECTRIC	PORTAGE	RAVENNA	UNKNOWN CREEK	DIESEL FUEL	25	GAL	0
93	GENERAL ELECTRIC	TRUMBULL	NILES	MOSQUITO CREEK	WASTE WATER	0	UNK	0
93	GENERAL ELECTRIC	TRUMBULL	NILES	MOSQUITO CREEK	WASTE WATER	0	UNK	0
93	GENERAL ELECTRIC	TRUMBULL	NILES	MOSQUITO CREEK	WASTE WATER	0	UNK	0
93	GENERAL ELECTRIC	TRUMBULL	NILES	MOSQUITO CREEK	WASTE WATER	0	UNK	0
93	GENERAL ELECTRIC	TRUMBULL	NILES	MOSQUITO CREEK	WASTE WATER	0	UNK	0
94	GENERAL ELECTRIC	TRUMBULL	NILES	MOSQUITO CREEK	WASTE WATER	0	UNK	0
92	GENERAL ELECTRIC	TRUMBULL	NILES	MOSQUITO CREEK	WASTE WATER	0	UNK	0
93	GENERAL ELECTRIC	TRUMBULL	NILES	MOSQUITO CREEK	WASTE HYDRAULIC OIL	20	GAL	0
90	GENERAL MOTORS / LORDSTO	TRUMBULL	LORDSTOWN	MEANDER CR RESV.	DIESEL FUEL	0	UNK	0
90	GENERAL MOTORS / LORDSTO	TRUMBULL	LORDSTOWN	STORM SEWER	WATER BASED PAINT	1100	GAL	0
91	GENERAL MOTORS / LORDSTO	TRUMBULL	LORDSTOWN	MUD CREEK TRIB	ANTIFREEZE	0	UNK	0
91	GENERAL MOTORS / LORDSTO	TRUMBULL	LORDSTOWN	STORM DRAIN	RINSEWATER	200	GAL	0
93	GENERAL MOTORS / PACKAR	TRUMBULL	WARREN	MAHONING RIVER	WASTE WATER	0	UNK	0
90	GENERAL MOTORS / PACKAR	TRUMBULL	WARREN	RED RUN CREEK	ANIMAL FAT & LUBRICATIN	15	GAL	0
93	GIRARD STP	TRUMBULL	GIRARD	LITTLE SQUAW CREEK	SEWAGE	0	UNK	0
90	GRANT STREET SCRAP OIL	TRUMBULL	NILES	MAHONING RIVER	MOTOR OIL	0	UNK	0
91	GREENWOOD ACCURA	MAHONING	BOARDMAN	STORM DRAIN	MOTOR OIL	0	UNK	0
93	GWIN CONTRACTING	COLUMBIAN	COLUMBIANA	UNKNOWN CREEK	FUEL OIL	15	GAL	0

YR	ENTITY	COUNTY	TWP_CITY	WATERWAY	MATERIAL_1	AMOUNT_1	UNITS_1	RECOVER_1
90	HARE EXPRESS/RUAN LEASIN	MAHONING	YOUNGSTOWN	STORM SEWER	DIESEL FUEL	70	GAL	0
90	HILLTOP LANDFILL	MAHONING	ELLSWORTH TW	PALMYRA LAKE-MEANDE RES.	VARIOUS MORGANIC	0	UNK	0
90	HOWELL INDUSTRIES	TRUMBULL	MASURY	SHENANGO RIVER	UNK WHITE STUFF	0	UNK	0
89	HUBBARD ELECTRIC DEPT	TRUMBULL	HUBBARD	UNKNWON	TRANSFORMER OIL	0	UNK	0
90	INDUSTRIAL CLEANING SERVI	TRUMBULL	GERRALD	CITY SEWER DRAINS	BLACK LIQUID	0	UNK	0
93	J & M TRUCKING	PORTAGE	ATWATER TWP	UNKNOWN CREEK	BRINE	0	UNK	0
90	J J COAS TRUCKING	TRUMBULL	WARREN	MAHONING RIVER	UNK	0	UNK	0
90	J L COATS DRILLING	MAHONING	AUSTONTOWN	SMALL CREEK	BRINE	0	UNK	0
89	J L COATS WELL SERVICE	MAHONING	ELLSWORTH TW	MEANDER CREEK	WATER PICKUP	0	NOS	0
91	JAMES DRILLING CORP	ASHTABULA	DORSETT TWP	WETLAND	CRUDE OIL	84	GAL	84
90	JEFFERY F. BATES	PORTAGE	GARRETTSVILLE	SILVER CREEK	WASTE OIL	275	GAL	0
89	JOHN KETTLER	TRUMBULL	BROCKFIELD	SPRINGFED CREEK	CONSTRUCTION WASTE	0	UNK	0
91	JOHN PITTMAN	MAHONING	POLAND TWP	STORM DITCH	RED STUFF	0	UNK	0
89	KAUFMAN DEPT STORE	MAHONING	BOARDMAN	UNKNOWN	ASBESTOS	0	UNK	0
90	KUNTZMAN TRUCKING	MAHONING	GOSHEN TWP	MILL CREEK	DIESEL FUEL	50	GAL	0
90	L T V STEEL,CAMPBELL	MAHONING	CAMPBELL	MAHONING RIVER	HYDROCARBON	0	UNK	0
89	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	MAHONING RIVER	WASTE WATER	3000	GAL	0
89	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	STORM SEWER	WASTE WATER	1500	GAL	100
89	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	STORM SEWER	WASTE WATER	500	GAL	0
90	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	MAHONING RIVER	WASTE WATER	0	UNK	0
90	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	MAHONING RIVER	WASTE WATER	2000	GAL	0
90	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	MAHONING RIVER	ABSORBENT OIL	100	GAL	0
90	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	MAHONING RIVER	DIRECT COOLING WATER	300	GAL	0
90	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	MAHONING RIVER	WASTE WATER	500	GAL	0
90	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	MAHONING RIVER TRIB	UNK HYDROCARBON	0	UNK	0
91	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	MAHONING RIVER	OIL	5	GAL	0
91	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	MAHONING RIVER	SULFURIC ACID	20	GAL	0
91	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	MAHONING RIVER	SULFURIC ACID	0	UNK	0
91	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	MAHONING RIVER TRIB	ACID WASTEWATER	109000	GAL	107000
91	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	STORM SEWER	TAR	110	GAL	0
91	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	UNKNOWN	CRUDE COAL TAR	200	GAL	0
92	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	MAHONING RIVER	WASTE WATER	2000	GAL	0
93	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	MAHONING RIVER	WASTE WATER	0	UNK	0
93	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	MAHONING RIVER	WASTE OIL	20	GAL	0
93	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	MAHONING RIVER	CONTAMINATED WASTEWA	500	GAL	0
93	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	MAHONING RIVER	OIL	1	GAL	0
93	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	MAHONING RIVER	OIL	0	UNK	0
94	L T V STEEL,WARREN COKE	TRUMBULL	WARREN	UNKNOWN CREEK	WASTE WATER	0	UNKNO	0
89	LAMAK PETROLEUM	PORTAGE	WINDOM	SILVER CREEK TRIB	CRUDE OIL	0	UNK	0
93	LARRY MURPHY TRUCKING	COLUMBIAN	SEBRING	DEER RUN	PETROLIUM OIL	1	UNK	0
91	LESKO RIDE CO	TRUMBULL	CORTLAND	STORM SEWER	HYDRAULIC FLUID	20	GAL	0
91	LOUISIANA PACIFIC CORP	MAHONING	BOARDMAN	MAHONING RIVER TRIB	HYDRAULIC OIL	0	UNK	0
92	LOWELVILLE ROD & GUN	MAHONING	LOWELVILLE	UNKNOWN CREEK	SOAPY STUFF	0	UNK	0
90	LYDEN OIL	MAHONING	CAMPBELL	MAHONING RIVER	GASLOINE	130	GAL	0
92	LYNN STRANG / STRANG'S JU	ASHTABULA	DORSET	UNKNOWN CREEK	SLUDGE	0	UNK	0
91	M & B OPERATING CO	PORTAGE	ROOTSTOWN TW	SWAMP	CRUDE OIL	2940	GAL	0
89	MAHONING CO SANI ENG DEP	MAHONING	AUSTINTOWN	UNNAMED CREEK	SEWAGE	0	UNK	0
91	MAHONING PAINT CORP	MAHONING	YOUNGSTOWN	MAHONING RIVER	PAINT	0	UNK	0
89	MAHONING VALLEY SANITARY	MAHONING	MINERAL RIDGE	MEANDER CREEK	SEWAGE	0	UNK	0
93	MAHONING VALLEY SANITARY	TRUMBULL	NILES	MEANDER CREEK	FLUOROSILICIC ACID	1500	GAL	0
89	MCGUIRES JUNKYARD	MAHONING	SPRINGFIELD TW	UNKNOWN	TRASH	0	UNK	0
92	MEANDER STP	TRUMBULL	MINERAL RIDGE	MEANDER CREEK	SEWAGE	0	UNK	0
90	MID STATE OIL FIELD	PORTAGE	GARRETSVILLE	UNKNOWN	NATURAL GAS	0	UNK	0
92	MILLER BROTHERS	ASHTABULA	CHERRY VALLEY	UNKNOWN CREEK	FERTILIZER 28%	10	GAL	0
94	MILLIKEN AUTOMOTIVE	TRUMBULL	WARREN	STORM SEWER	KEROSENE	0	UNK	0

NTITY	COUNTY	TWP_CITY	WATERWAY	MATERIAL_1	AMOUNT_1	UNITS_1	RECOVER_1
IOTHER NATURE	STARK	ALLIANCE	DEER CREEK	UNKNOWN	0	UNK	0
IOTHER NATURE	ASHTABULA	ANDOVER TWP	BLOCK CREEK TRIB	ALGAE	0	UNK	0
IOTHER NATURE	MAHONING	LAKE MILTON	LAKE MILTON	ALGAE	0	UNK	0
IOTHER NATURE	MAHONING	YOUNGSTOWN	PUBLIC POND	ALGAE	0	NOS	0
IOTHER NATURE	PORTAGE	ROOTSTOWN	POND	OIL	0	UNK	0
IOTHER NATURE	MAHONING	LAKE MILTON	LAKE MILTON	NO SPILL	0	NOS	0
IOTOR FREIGHT I NC.	MAHONING	MILTON TWP	LAKE MILTON	DIESEL FUEL	115	GAL	0
IR ART TILTON	TRUMBULL	LEAVITTSBURG	UNKNOWN CREEK	FUEL OIL	5	GAL	0
IR BARRY BAER	TRUMBULL	NEWTON FALLS	UNKNOWN	OIL	0	UNK	0
IR BENOWSKI	PORTAGE	HIRAM	EAGLE CREEK	MANURE	0	UNK	0
IR CHARLES SMITH	MAHONING	CRAIG BEACH	LAKE MILTON	OIL	0	UNK	0
IR DENZIL SNYDER	TRUMBULL	CORTLAND	MOSQUITO CREEK TRIB	FUEL OIL	275	GAL	100
IR HERMIT DEAN JR	PORTAGE	WINDOM TWP	UNKNOWN CREEK	UNK	0	UNK	0
IR JOHN GORAL	TRUMBULL	BRACEVILLE TW	MAHONING RIVER TRIB	PAINT	5	GAL	0
IR JOHN PITTMAN	MAHONING	POLAND	STORM DITCH	RED STUFF	0	UNK	0
IR LAURA HUTCHINS	TRUMBULL	CHAMPION TWP	STORM SEWER-MAHONING RIV	HEATING OIL	20	GAL	15
IR LEO SORICE	MAHONING	BOARDMAN	MILL CREEK	DIESEL FUEL	0	UNK	0
IR LLOYD SHERIDAN	MAHONING	YOUNGSTOWN	UNKNOWN CREEK	CONCRETE	0	UNK	0
IR NOVAK	TRUMBULL	HARTFORD	WETLANDS	ASPHALT	0	UNK	0
IR PETERS	COLUMBIAN	KNOX TWP	UNKNOWN CREEK	BLACK OIL STUFF	0	UNK	0
IR RANDY SMILEY	MAHONING	ELLSWORTH	UNKNOWN	MOTOR OIL	70	GAL	0
IR RANKIN	PORTAGE	RAVENNA TWP	UNKNOWN CREEK	TRASH	0	UNK	0
IR RICHARD GRUND	PORTAGE	RAVENNA	HINKLEY CREEK	FUEL OIL	100	GAL	0
IR ROBERT COOK	MAHONING	YOUNGSTOWN	SEWER	WASTE OIL	0	UNK	0
IR ROY CARSON	MAHONING	BERLIN TWP	MILL CREEK	FUEL OIL	0	UNK	0
IR SAM RAFIDE	MAHONING	YOUNGSTOWN	STORM SEWER	OILY STUFF	5	GAL	0
IR VANDERHOVER	PORTAGE	NELSON	MAHONING RIVER TRIB	DRUMS	0	UNK	0
IR VINCE HAROLD	STARK	LEXINGTON	DEER CREEK	DIESEL FUEL	5	GAL	3
IR ZALID	PORTAGE	WINDOM	S F CREEK	WASTEWATER	0	UNK	0
IR. WILBER HOPTON	STARK	LEXINGTON TWP	UNKNOWN	FUEL OIL	144	GAL	0
IS IRENE WORK	PORTAGE	NELSON TWP	UNKNOWN CREEK	GARBAGE	0	UNK	0
ULTICLEAR SERVICE	MAHONING	YOUNGSTOWN	STORM SEWER	UNKNOWN	0	UNK	0
UNSON TRANSPORTATION	MAHONING	YOUNGSTOWN	STORM SEWER	DIESEL FUEL	100	GAL	0
URPHY TRUCKING CO	COLUMBIAN	DAMASCUS	MAHONING RIVER	BLUE GREEN STUFF	0	UNK	0
OBLE OIL	PORTAGE	EDINBURG TWP	UNNAMED CREEK	CRUDE OIL	30	GAL	0
ORTH AMERICAN VAN LINES	PORTAGE	PALMIRE TWP	UNKNOWN	DIESEL FUEL	100	GAL	0
ORTH CANTON TRANSFER	MAHONING	YOUNGSTOWN	UNKNOWN	DIESEL FUEL	1200	GAL	1200
ORTH STAR STEEL	MAHONING	YOUNGSTOWN	MAHONING RIVER	WASTE WATER	93000	GAL	0
ORTH STAR STEEL	MAHONING	YOUNGSTOWN	MAHONING RIVER	OIL & GREASE	0	GAL	0
ORTH STAR STEEL	MAHONING	YOUNGSTOWN	MAHONING RIVER	WASTE WATER	139000	GAL	0
ORTH STAR STEEL	MAHONING	YOUNGSTOWN	MAHONING RIVER	OIL & GREASE	0	UNK	0
ORTH STAR STEEL	MAHONING	YOUNGSTOWN	MAHONING RIVER	WASTE WATER	164000	GAL	0
ORTH STAR STEEL	MAHONING	YOUNGSTOWN	MAHONING RIVER	WASTE WATER	0	UNK	0
ORTH STAR STEEL	MAHONING	YOUNGSTOWN	MAHONING RIVER	WASTE WATER	0	UNK	0
ORTH STAR STEEL	MAHONING	YOUNGSTOWN	MAHONING RIVER	MOIL/GREASE	300	GAL	0
ORTH STAR STEEL	MAHONING	YOUNGSTOWN	MAHONING RIVER	T.S.S. WASTE WATER	186000	GAL	0
ORTH STAR STEEL	MAHONING	YOUNGSTOWN	MAHONING RIVER	HYDROCARBON	0	UNK	0
ORTH STAR STEEL	MAHONING	YOUNGSTOWN	MAHONING RIVER	WASTEWATER	136000	GAL	0
ORTH STAR STEEL	MAHONING	YOUNGSTOWN	MAHONING RIVER	OIL AND GREASE	0	UNK	0
ORTH STAR STEEL	MAHONING	YOUNGSTOWN	MAHONING RIVER	OIL AND GREASE	0	UNK	0
ORTH STAR STEEL	MAHONING	YOUNGSTOWN	MAHONING RIVER	WASTE WATER	225000	GAL	0
ORTH STAR STEEL	MAHONING	YOUNGSTOWN	MAHONING RIVER	OIL	24	KGS	0
ORTH STAR STEEL	MAHONING	YOUNGSTOWN	MAHONING RIVER	OIL	0	UNK	0
ORTH STAR STEEL	MAHONING	YOUNGSTOWN	MAHONING RIVER	OIL	41	KGS	0
ORTH STAR STEEL	MAHONING	YOUNGSTOWN	MAHONING RIVER	OIL	18	KGM	0

YR	ENTITY	COUNTY	TWP_CITY	WATERWAY	MATERIAL_1	AMOUNT_1	UNITS_1	RECOVER_1
89	NOVAK AIRCRAFT MAINTENAN	PORTAGE	RAVENNA	UNKNWON	OIL	3	GAL	0
89	O & P FUEL OIL CO	ASHTABULA	RICHMAN	UNKNOWN	FUEL OIL	275	GAL	50
89	ODOT	MAHONING	CANFIELD	STORM SEWER	DIESEL FUEL	1600	GAL	0
90	ODOT	TRUMBULL	CORTLAND	STORM SEWER	DIESEL FUEL	0	UNK	0
93	ODOT	MAHONING	CANFIELD	MEANDER CREEK	ASPHALT EMULSION	500	GAL	0
93	ODOT	TRUMBULL	HUBBARD	UNKNOWN CREEK	WASTE OIL	0	UNK	0
92	OHIO DEPT NATURAL RESOUR	PORTAGE	DEERFIELD	BERLIN RESERVIOR	FUEL OIL	350	GAL	0
89	OHIO EDISON	TRUMBULL	WEATHERSFIELD	MAHONING RIVER	SOOT	0	UNK	0
90	OHIO EDISON	TRUMBULL	NILES	MAHONING RIVER	WASTE HYDROCHLORIC AC	150	GAL	0
91	OHIO EDISON	TRUMBULL	NILES	MAHONING RIVER	ASH	540	CFT	0
93	OHIO EDISON	TRUMBULL	NILES	MAHONING RIVER	WASTE WATER	0	UNK	0
93	OHIO EDISON	TRUMBULL	NILES	MAHONING RIVER	WASTE WATER	0	UNK	0
93	OHIO EDISON	TRUMBULL	NILES	MAHONING RIVER	WASTE WATER	0	UNK	0
93	OHIO EDISON	TRUMBULL	NILES	MAHONING RIVER	WASTEWATER	0	UNK	0
93	OHIO EDISON	TRUMBULL	NILES	MAHONING RIVER	WASTEWATER	0	UNK	0
93	OHIO EDISON	TRUMBULL	NILES	MAHONING RIVER	WASTEWATER	0	UNK	0
91	OHIO EDISON	TRUMBULL	NILES	MAHONING RIVER	ACID	215	GAL	0
89	OHIO EDISON ELECTRIC CO	TRUMBULL	WARREN	MAHONING RIVER	GASOLINE	600	GAL	0
92	OHIO SCRAP & IRON	TRUMBULL	WARREN	MAHONING RIVER	HYDRAULIC OIL	0	UNK	0
91	OHIO WATER SERVICE	TRUMBULL	GIRARD	SQUAW CREEK TRIB	ODOR OF SULFUR	0	UNK	0
90	OLD SHEET & TUB MILL	MAHONING	STRUTHERS	MAHONING RIVER	OIL	0	UNK	0
91	ORCHARD ESTATES TRAILER	PORTAGE	RAVENNA	PUBLIC WATER SUPPLY	RUSTY ORANGE WATER	0	UNK	0
89	ORION ENERGY	PORTAGE	ATWATER TWP	WATER SUPPLY	CRUDE OIL	3300	GAL	3200
92	ORION PETROLEUM CORP	PORTAGE	ATWATER TWP	DEER CREEK	CRUDE OIL	3570	GAL	2600
93	P N L HEAT TREATMENT	MAHONING	YOUNGSTOWN	STORM SEWER	OIL	0	UNK	0
93	PACKARD ELECTRIC CO	TRUMBULL	VIENNA TWP	SPRING RUN	GREEN STUFF	0	UNK	0
90	PALUMBO CLEANERS	MAHONING	CANFIELD	STORM SEWER	SUDS	0	UNK	0
91	PANDA TRUCKING CO	TRUMBULL	WARREN	UNKNOWN CREEK	WATER PICKUP	0	NOS	0
89	PAUL BIGELOW & SONS	MAHONING	MILTON TWP	UNKNWON	SEWAGE	0	UNK	0
93	PENTECH ENTERPRISES	TRUMBULL	FOWLER TWP	UNKNOWN CREEK	DIESEL FUEL	150	GAL	0
92	PLY-TRIM CORP	MAHONING	AUSTINTOWN	MAHONING RIVER TRIB	WATER BASED LATEX PRIM	50	GAL	0
93	PORTAGE COUNTY ENGINEER	PORTAGE	RAVENNA	SILVER CREEK	HYDRAULIC OIL	10	GAL	0
90	PORTS PETROLEUM	MAHONING	AUSTINTOWN	UNKNOWN	DIESEL FUEL	0	UNK	0
89	PRODEX INC	PORTAGE	RAVENNA	HINKLEY CREEK	WASTE WATER	0	UNK	0
90	PSK STEEL	TRUMBULL	HUBBARD	STORM SEWER	CUTTING OIL	0	UNK	0
92	PUTNAM TRANSFER	PORTAGE	RAVENNA	STORM SEWER	GASOLINE	5	GAL	0
92	QUAKER STATE OIL / CRUDE O	PORTAGE	ATWATER TWP	UNKNOWN	OIL	714	GAL	672
89	QUAKER STATE OIL CO	ASHTABULA	WAYNE TWP	UNKNOWN	CRUDE OIL	55	GAL	0
91	QUAKER STATE OIL CO	TRUMBULL	WEATHERSFIELD	UNKNOWN	CRUDE OIL	42	GAL	0
92	R KUNZMAN INC	STARK	ALLIANCE	STORM SEWER	DIESEL	50	GAL	0
90	R M I TITANIUM	TRUMBULL	NILES	MAHONING RIVER TRIB	PCB CONTAMINATED WAST	0	UNK	0
92	R M I TITANIUM	TRUMBULL	NILES	MAHONING RIVER	WASTE WATER	0	UNK	0
93	R M I TITANIUM	TRUMBULL	NILES	MAHONING RIVER	OIL	0	UNK	0
91	R M I TITANIUM	TRUMBULL	NILES	HOLDING POND	HYDROFLOURIC ACID	15	GAL	0
93	R M I TITANIUM	TRUMBULL	NILES	MAHONING RIVER	OIL	25	GAL	0
94	R M I TITANIUM	TRUMBULL	NILES	MAHONING RIVER	WASTE WATER	0	UNK	0
89	RAVENNA ARMY AMMUNITION	PORTAGE	WINDHAM	UNKNOWN	TNT WASTE WATER	350	GAL	0
92	RAVENNA ARMY AMMUNITION	PORTAGE	RAVENNA	SAND CREEK TRIB	WASTE WATER	180000	GAL	0
92	RAVENNA CITY GARAGE	PORTAGE	RAVENNA	SEWER	GASOLINE	25	GAL	20
89	RAY PANDER TRUCKING	MAHONING	CANFIELD	SMALL CREEK	BRINE	0	UNK	0
93	RAYMOND ANDERSON FARM	MAHONING	CANFIELD	INDIAN RUN	PESTICIDES	0	UNK	0
91	REGAL TRANSPORTATION	TRUMBULL	NILES	UNKNOWN	DIESEL FUEL	0	UNK	0
89	REMMANT ROOM	TRUMBULL	BROOKFIELD TW	UNKNOWN	CHEMICALS	0	UNK	0
91	RIVER BEND TRANSPORT/OTR	MAHONING	AUSTIN	UNKNOWN	DIESEL FUEL	300	GAL	0
93	RIVERSIDE AUTO CENTER	MAHONING	ALLIANCE	MAHONING RIVER	JUNK	0	UNK	0

YR	ENTITY	COUNTY	TWP_CITY	WATERWAY	MATERIAL_1	AMOUNT_1	UNITS_1	RECOVER_1
92	ROBERTSON HEATING & SUPP	STARK	ALLIANCE	STORM SEWER	DIESEL FUEL	105	GAL	90
89	ROLAND BROTHERS WAREHO	MAHONING	YOUNGSTOWN	FOUR MILE RUN	SMOKE	0	UNK	0
90	RYDER TRUCK RENTAL	MAHONING	NEW MIDDLETON	UNKNOWN	DIESEL FUEL	50	GAL	0
91	SAFETY KLEEN	TRUMBULL	HUBBARD	STORM SEWER	MINERAL SPIRITS	9	GAL	0
89	SAUNDERS CO	PORTAGE	RAVENNA	UNKNOWN	DIESEL FUEL	0	UNK	0
90	SAUNDERS EXCAVATING	PORTAGE	RAVENNA	UNKNWON	OIL	0	UNK	0
92	SAVAET SERVICE INC	MAHONING	N LIMA	UNKNOWN CREEK	OIL	0	UNK	0
92	SAVEAT OIL CO	MAHONING	NORTH LIMA	UNKNOWN	NO SPILL	0	UNK	0
89	SCHAEFER EQUIPMENT INC	TRUMBULL	WARREN	RED RUN CREEK	WASTE WATER	0	UNK	0
89	SCHAEFER EQUIPMENT INC	TRUMBULL	WARREN	RED RUN CREEK		0	UNK	0
91	SCHAEFER EQUIPMENT INC	TRUMBULL	WARREN	RED RUN CREEK	SUSPENDED SOLIDS	0	UNK	0
91	SCHAEFER EQUIPMENT INC	TRUMBULL	WARREN	RED RUN CREEK	OIL (13 MG/L)	0	UNK	0
91	SCHAEFER EQUIPMENT INC	TRUMBULL	WARREN	RED RUN CREEK	OIL	28	PPM	0
91	SCHAEFER EQUIPMENT INC	TRUMBULL	WARREN	RED RUN CREEK	OIL	0	UNK	0
91	SCHAEFER EQUIPMENT INC	TRUMBULL	WARREN	RED RUN CREEK	WASTE WATER	0	UNK	0
92	SCHAEFER EQUIPMENT INC	TRUMBULL	WARREN	RED RUN CREEK	WASTE WATER	0	UNK	0
92	SCHAEFER EQUIPMENT INC	TRUMBULL	WARREN	RED RUN CREEK	OIL	0	UNK	0
92	SCHAEFER EQUIPMENT INC	TRUMBULL	WARREN	RED RUN CREEK	WASTE WATER	0	UNK	0
93	SCHAEFER EQUIPMENT INC	TRUMBULL	WARREN	RED RUN CREEK	WASTE WATER	0	UNK	0
91	SCHAEFFER METAL PRODUCT	PORTAGE	RAVENNA	STORM SEWER	WASTE CHEMICALS	0	UNK	0
89	SEBRING CHURCH OF CHRIST	MAHONING	SEBRING	PRIVATE POND	FISHKILL	80	ITM	0
90	SERVO CLEAN	MAHONING	YOUNGSTOWN	STORM SEWER	UNK BLACK LIQUID & SLUD	0	UNK	0
90	SHADYBROOK MOBIL HOME P	MAHONING	BOARDMAN	STORM SEWER-MILL CREEK	FUEL OIL	200	GAL	0
91	SHAHEEN PLUMBING & HEATI	STARK	ALLIANCE	STORM SEWER	WASTE OIL	40	GAL	40
91	SODA CONSTRUCTION	STARK	LEXINGTON TWP	BERLIN RESERVOIR	UNKNOWN	110	GAL	0
90	SOUTHERN RADIATOR	MAHONING	YOUNGSTOWN	SEWER	ANTIFREEZE	0	UNK	0
91	SOUTHWEST MOTOR FREIGHT	MAHONING	AUSTINTOWN	MAHONING RIVER TRIB	DIESEL FUEL	150	GAL	0
93	SPARKS TUNE UP	TRUMBULL	WARREN	STORM SEWER	OIL	0	UNK	0
92	STANDARD LAFARGE	MAHONING	YOUNGSTOWN	UNKNOWN	GREASE	0	UNK	0
89	STAR ROOFING/COVELLI PRO	TRUMBULL	NILES	MOSQUITO CREEK TRIB	TAR	0	UNK	0
91	STELL CITY	MAHONING	AUSTINTOWN TW	STORM SEWER	OIL	0	UNK	0
89	STRUTHERS AUTO SERVICE	MAHONING	STRUTHERS	MAHONING RIVER TRIB	GASOLINE	15	GAL	0
90	STRUTHERS CSO	MAHONING	STRUTHERS	YELLOW CREEK	SEWAGE	0	UNK	0
92	STRUTHERS STREET DEPT	MAHONING	STRUTHERS	MAHONING RIVER	METAL SHAVINGS	0	UNK	0
89	SUMMIT NATIONAL	PORTAGE	DEERFIELD	BERLIN RESERVOIR	WASTE WATER	0	UNK	0
90	T & W FORGE INC.	STARK	ALLIANCE	UNKNOWN	FUEL OIL	300	GAL	0
93	THERM-O-LINK	PORTAGE	GARRETTSVILLE	SILVER CREEK	YELLOW MATERIAL	0	UNK	0
90	THERMAL TECH INC.	TRUMBULL		MAHONING RIVER	ORANGE STUFF	0	UNK	0
90	THERMATEX	MAHONING	NEWTON FALLS	MAHONING RIVER	SOLUBLE OIL	0	UNK	0
89	TIM WEAVER	MAHONING	POLAND	EVANS LAKE	TAR OIL	0	UNK	0
90	TOM'S SEWER & DRAINS	TRUMBULL	GERRAD	MAHONING RIVER	SEWAGE	0	UNK	0
90	TOP LINE	TRUMBULL	LORDSTOWN	UNKNOWN	DIESEL FUEL	100	GAL	0
93	TRI STATE MOTOR TRANSIT C	PORTAGE	CHARLESTON TW	W BRANCH RESERVOIR	DIESEL FUEL	50	GAL	40
89	TRUCK STOPS OF AMERICA	MAHONING	LIMA	UNKNOWN	DIESEL FUEL	0	UNK	0
89	TRUCK STOPS OF AMERICA	MAHONING	NORTH LIMA	UNKNOWN	DIESEL FUEL	0	UNK	0
90	TRUCK STOPS OF AMERICA	MAHONING	N. LIMA	MILL CREEK	GASOLINE	0	UNK	0
89	TRUCK WASH	MAHONING	LIMA	UNKNOWN CREEK	SOAP & DIRT	0	UNK	0
93	TRUE GREEN CHEMICALS	MAHONING	N JACKSON TWP	PRIVATE POND	FERTILIZER	94	GAL	0
93	TRUMBULL MEMORIAL HOSPIT	TRUMBULL	WARREN	MAHONING RIVER	DIESEL FUEL	640	GAL	0
89	TURKEY FARM	PORTAGE	NELSON TWP	COMP CREEK	FISHKILL	200	ITM	0
91	UNITED EXCAVATING AVE	MAHONING	YOUNGSTOWN	STORM SEWER-MAHONING RIV	OIL	0	UNK	0
90	UNIVERSAL TRUCK PLAZA/MA	MAHONING	YOUNGSTOWN	UNNAMED CREEK	DIESEL FUEL	200	GAL	0
89	UNK	MAHONING	BOARDMAN	MILL CREEK	ILLEGAL DUMPING	0	UNK	0
89	UNK	MAHONING	SMITH TWP	UNKNOWN	JUNK, TRASH	0	UNK	0
89	UNK	MAHONING	GIRARD	UNKNOWN	GREEN MATERIAL	0	UNK	0

YR	ENTITY	COUNTY	TWP_CITY	WATERWAY	MATERIAL_1	AMOUNT_1	UNITS_1	RECOVER_1
90	UNK	MAHONING	N. JACKSON	MEANDER CK RESV.	UNK	0	UNK	0
90	UNK	MAHONING	YOUNGSTOWN	STORM SEWER	UNK	10	DRM	0
90	UNK	MAHONING	YOUNGSTOWN	STORM SEWER	UNK	0	UNK	0
90	UNK	MAHONING	BOARDMAN	STORM SEWER	CUTTING OIL	0	UNK	0
90	UNK	MAHONING	N. LIMA	UNNAMED CREEK	UNK	1	DRM	0
90	UNK	PORTAGE	GARRETTSVILLE	SILVER CREEK	DIESEL FUEL	300	GAL	0
90	UNK	PORTAGE	ATWATER	STORM SEWER	OILY SUBSTANCE	0	UNK	0
90	UNK	PORTAGE	PALMIRA TWP	UNKNOWN	DIESEL FUEL	300	GAL	0
90	UNK	PORTAGE	GARRETSVILLE	UNKNOWN	UNK	110	GAL	0
90	UNK	PORTAGE	PALMYRA TWP	UNKNOWN CREEK	OIL	0	UNK	0
90	UNK	TRUMBULL	LIBERTY TWP	LITTLE SQUAW CREEK	BLACK WATER-DRIVEWAY	0	UNK	0
90	UNK	TRUMBULL	NEWTON FALLS	MAHONING RIVER TRIB	OIL	0	UNK	0
90	UNK	TRUMBULL	CHAMPION	STORM SEWER	UNK	0	UNK	0
89	UNKNOWN	MAHONING	YOUNGSTOWN	BEAR CREEK	CUTTING OIL	0	UNK	0
89	UNKNOWN	MAHONING	AUSTINTOWN TW	BEAR CREEK	MILKY WHITE STUFF	0	UNK	0
89	UNKNOWN	MAHONING	BROADMAN TWP	HUNTERS CREEK	ORANGE STUFF	0	UNK	0
89	UNKNOWN	MAHONING	YOUNGSTOWN	LAKE GLACIER	SEWAGE	0	UNK	0
89	UNKNOWN	MAHONING	AUSTINTOWN TW	MEANDER CK RESV.	UNKNOWN	0	UNK	0
89	UNKNOWN	MAHONING	ELLSWORTH	MEANDER CREEK	CRUDE OIL	0	UNK	0
89	UNKNOWN	MAHONING	AUTINTOWN	UNKNOWN	WHITE STUFF	0	UNK	0
89	UNKNOWN	MAHONING	GASHEN	UNKNOWN	BLACK STUFF	0	UNK	0
89	UNKNOWN	MAHONING	BEAVER TWP	UNKNOWN	OIL	0	UNK	0
89	UNKNOWN	MAHONING	BOARDMAN	UNKNOWN CREEK	BRINE	0	UNK	0
89	UNKNOWN	MAHONING	NORTH LIMA	YELLOW CREEK	HYDROCARBON	0	UNK	0
89	UNKNOWN	MAHONING	POLAND	YELLOW CREEK	TRASH	0	UNK	0
89	UNKNOWN	PORTAGE	PALMYRA TWP	POLE CREEK	FISHKILL	0	UNK	0
89	UNKNOWN	PORTAGE	RAVENNA	POND	FISHKILL	100	ITM	0
89	UNKNOWN	PORTAGE	GARRETSVILLE	SILVER CREEK	DIESEL FUEL	0	UNK	0
89	UNKNOWN	PORTAGE	ATWATER TWP	UNKNOWN	FUEL OIL	400	GAL	0
89	UNKNOWN	PORTAGE	ATWATER TWP	UNKNOWN	OIL	0	UNK	0
89	UNKNOWN	TRUMBULL	WARREN	MAHONING RIVER	FOAM	0	UNK	0
89	UNKNOWN	TRUMBULL	WARREN	MOSQUITO CREEK	BRIGHT BLUE SUBSTANCE	0	UNK	0
89	UNKNOWN	TRUMBULL	BROOKFIELD TW	UNKNOWN	CHEMICAL	0	UNK	0
89	UNKNOWN	TRUMBULL	HUBBARD TWP	UNKNOWN	ASBESTOS	0	UNK	0
90	UNKNOWN	COLUMBIAN	BELOIT	WESTVILLE LAKE RESERVIOR	DIESEL FUEL	100	GAL	0
90	UNKNOWN	MAHONING	YOUNGSTOWN	BEAR CREEK	UNK WHITE STUFF	0	UNK	0
90	UNKNOWN	MAHONING	POLEN	BURGESS LAKE	RAW SEWAGE	0	UNK	0
90	UNKNOWN	MAHONING	AUSTINTOWN	HOLDING POND	DIESEL FUEL	160	GAL	0
90	UNKNOWN	MAHONING	STROTHERS	MAHONING RIVER	OILY SUBSTANCE	0	UNK	0
90	UNKNOWN	MAHONING	CANFIELD	MILL CREEK TRIB	GREEN STUFF	0	UNK	0
90	UNKNOWN	MAHONING	NEWTON FALLS	POND	OIL SHEEN	0	UNK	0
90	UNKNOWN	MAHONING	YOUNGSTOWN	UNKNOWN	BRINE	0	UNK	0
90	UNKNOWN	PORTAGE	DERRFIELD	UNNAMED CREEK	RED WATER	0	UNK	0
90	UNKNOWN	PORTAGE		UNNAMED CREEK	BRINE	0	UNK	0
90	UNKNOWN	STARK	ALLIANCE	UNKNOWN	BRINE	0	UNK	0
90	UNKNOWN	STARK	ALLIANCE	UNKNOWN	CRUDE OIL	0	UNK	0
90	UNKNOWN	STARK	ALLIANCE	UNKNOWN	DIESEL FUEL	0	UNK	0
90	UNKNOWN	TRUMBULL	NILES	MAHONING RIVER	BROUN STUFF	0	UNK	0
90	UNKNOWN	TRUMBULL	NEWTON FALLS	MAHONING RIVER	FISH KILL	0	UNK	0
90	UNKNOWN	TRUMBULL	CHAMPION TWP	MAHONING RIVER TRIB	OIL	0	UNK	0
90	UNKNOWN	TRUMBULL	BAZETTA	MOSQUITO CREEK	DIESEL FUEL	0	UNK	0
90	UNKNOWN	TRUMBULL	WARREN	UNKNOWN	ABANDONED DRUMS	2	ITM	2
90	UNKNOWN	TRUMBULL	NEWTON FALLS	W B MAHONING RIVER	WHITE FOAM	0	UNK	0
90	UNKNOWN	TRUMBULL C	LIBERTY TWP	LIBERTY GERARD LAKE	FOAM	0	UNK	0
91	UNKNOWN	ASHTABULA	WAYNE TWP	POND	UNIDENTIFIED OIL	1	GAL	1

YR	ENTITY	COUNTY	TWP_CITY	WATERWAY	MATERIAL_1	AMOUNT_1	UNITS_1	RECOVER_1
91	UNKNOWN	MAHONING	YOUNGSTOWN	BEAR CREEK	OIL	0	UNK	0
91	UNKNOWN	MAHONING	YOUNGSTOWN	MILL CREEK TRIB	ANTI FREEZE	0	UNK	0
91	UNKNOWN	MAHONING	AUSTINTOWN TW	STORM SEWER-MILL CREEK TR	WHITE SOLUBLE OIL	50	GAL	0
91	UNKNOWN	MAHONING	BEAVER TWP	TURKEY CREEK TRIB	BLUE DYE	0	UNK	0
91	UNKNOWN	MAHONING	POLAND	UNKNOWN	DYE	0	UNK	0
91	UNKNOWN	TRUMBULL	NEWTON FALLS	MAHONING RIVER	CONCRETE	0	UNK	0
91	UNKNOWN	TRUMBULL	WARREN	MAHONING RIVER TRIB	OIL	0	UNK	0
91	UNKNOWN	TRUMBULL	ALLEN	MOSQUITO CREEK TRIB	SEWAGE	0	UNK	0
91	UNKNOWN	TRUMBULL	HOLLAND TWP	MOSQUITO CREEK TRIB	GREEN STUFF	0	UNK	0
91	UNKNOWN	TRUMBULL	WARREN	PARK POND	VOLVOX AQUATIC LIFE	0	UNK	0
91	UNKNOWN	TRUMBULL	HOWLAND TWP	POND	ABANDONED DRUM	0	UNK	0
91	UNKNOWN	TRUMBULL	BROOKFIELD	STORM DITCH	OIL	0	UNK	0
92	UNKNOWN	ASHTABULA	WILLIAMSFIELD	UNKNOWN CREEK	DIESEL FUEL	0	UNK	0
92	UNKNOWN	MAHONING	YOUNGSTOWN	BEAR CREEK	WHITE STUFF	0	UNK	0
92	UNKNOWN	MAHONING	BERLIN CENTER	BERLIN RESERVOIR	ALGAE	0	UNK	0
92	UNKNOWN	MAHONING	YOUNGSTOWN	LAKE NEWPORT	MILKY STUFF	0	UNK	0
92	UNKNOWN	MAHONING	YOUNGSTOWN	LAKE NEWPORT	OIL	0	UNK	0
92	UNKNOWN	MAHONING	YOUNGSTOWN	MAHONING RIVER	GREEN STUFF	0	UNK	0
92	UNKNOWN	MAHONING	AUSTINTOWN	MEANDER CREEK	DIESEL FUEL	0	UNK	0
92	UNKNOWN	MAHONING	BOARDMEN	MILL CREEK	COLOR	0	UNK	0
92	UNKNOWN	MAHONING	BOARDMAN	MILL CREEK	UNKNOWN	0	UNK	0
92	UNKNOWN	MAHONING	BOARDMAN	MILL CREEK	IRON	0	UNK	0
92	UNKNOWN	MAHONING	CANFIELD	MILL CREEK	HEATING OIL	0	UNK	0
92	UNKNOWN	MAHONING	CANFIELD	MILL CREEK	OIL	0	UNK	0
92	UNKNOWN	MAHONING	CANFIELD	SAWMILL RUN CREEK TRIB	UNKNOWN STUFF	0	UNK	0
92	UNKNOWN	MAHONING	BOARDMAN TWP	STORM SEWER	GASOLINE	20	GAL	0
92	UNKNOWN	MAHONING	BEAVER TWP	STRIP MINE LAKE OUTLET	UNKNOWN STUFF	0	UNK	0
92	UNKNOWN	MAHONING	COITSVILLE TWP	UNKNOWN	CRUSTY SHEEN	0	UNK	0
92	UNKNOWN	MAHONING	YOUNGSTOWN	UNKNOWN	DEAD FISH	100	ITM	0
92	UNKNOWN	PORTAGE	DEERFIELD TWP	BERLIN RESERVOIR	DRUM	1	ITM	0
92	UNKNOWN	PORTAGE	EDINBURGH TWP	UNKNOWN CREEK	BRINE	0	UNK	0
92	UNKNOWN	STARK	LEXINGTON	BERLIN RESERVOIR	BLACK FOAMY STUFF	0	UNK	0
92	UNKNOWN	TRUMBULL	NEWTON FALLS	E B MAHONING RIVER	IRON	0	UNK	0
92	UNKNOWN	TRUMBULL	NEWTON FALLS	MAHONING RIVER	SEWAGE	0	UNK	0
92	UNKNOWN	TRUMBULL	WARREN	MAHONING RIVER	OIL	0	UNK	0
92	UNKNOWN	TRUMBULL	WARREN	RED LAKE	BLUE STUFF	0	UNK	0
92	UNKNOWN	TRUMBULL	GIRARD	UNKNOWN	KEROSENE	0	UNK	0
92	UNKNOWN	TRUMBULL	BRACEVILLE	UNKNOWN	OIL	25	GAL	0
92	UNKNOWN	TRUMBULL	NEWTON FALLS	W B MAHONING RIVER TRIB	IRON	0	UNK	0
93	UNKNOWN	COLUMBIAN	BUTLER TWP	STORM SEWER-PRIVATE POND	FISH KILL	24	ITM	0
93	UNKNOWN	MAHONING	YOUNGSTOWN	CASCADE RAVINE	SOAP	0	UNK	0
93	UNKNOWN	MAHONING	STRUTHERS	MAHONING RIVER	OIL	0	UNK	0
93	UNKNOWN	MAHONING	GOSHEN	UNKNOWN	DIESEL FUEL	0	UNK	0
93	UNKNOWN	MAHONING	SMITH TWP	UNKNOWN CREEK	OIL	0	UNK	0
93	UNKNOWN	MAHONING	AUSTINTOWN	UNKNOWN CREEK	OIL	4	GAL	0
93	UNKNOWN	MAHONING	POLAND	YELLOW CREEK	OIL	0	UNK	0
93	UNKNOWN	STARK	ALLIANCE	UNKNOWN CREEK	OIL	0	UNK	0
93	UNKNOWN	TRUMBULL	CHAMPION	MAHONING RIVER	FLUORESCEIN DYE	0	UNK	0
93	UNKNOWN	TRUMBULL	WEATHERSFIELD	MAHONING RIVER TRIB	FOAM	0	UNK	0
93	UNKNOWN	TRUMBULL		UNKNOWN	PLASTIC	0	UNK	0
90	UNKNOWN STP	PORTAGE	LAKE MILTON	LAKE MILTON	RAW SLUDGE	0	UNK	0
92	UNKNOWN*SUSPECTED*NEW	MAHONING	AUSTINTOWN	UNKNOWN CREEK	OIL	30	GAL	0
91	UNOCAL REFINING & MARKETI	MAHONING	AUSTINTOWN TW	SULFUR RUN TRIB	DIESEL FUEL	0	UNK	0
90	UNOCAL/YOUNGSTOWN 76	MAHONING	YOUNGSTOWN	UNKNOWN	DIESEL FUEL	150	GAL	0
92	VALVOLINE INSTANT OIL CHAN	MAHONING	AUSTINTOWN	UNKNOWN CREEK	ANTI-FREEZE	0	UNK	0

YR	ENTITY	COUNTY	TWP_CITY	WATERWAY	MATERIAL_1	AMOUNT_1	UNITS_1	RECOVER_1
93	VALVOLINE OIL CHANGE	MAHONING	BOARDMAN	STORM SEWER	OIL	500	GAL	0
90	VARIOUS JUNKYARDS	STARK	ALLIANCE	MAHONING RIVER	TRASH & JUNK	0	UNK	0
90	VERNAL PAVING	MAHONING	N.LIMA	UNKNOWN	OIL	0	UNK	0
90	VERNON SAND & GRAVEL	TRUMBULL	VERNON	ENTIRE WATERTABLE	UNCENERATED ASH	0	UNK	0
92	VERNON TWP TRUSTEES	TRUMBULL	VERNON TWP	PYMATUNING CREEK	DIESEL FUEL	200	GAL	200
90	VIKING RESOURCES	PORTAGE	ATWATER TWP	UNNAMED CREEK	CRUDE OIL	0	UNK	0
93	VIKING RESOURCES CORP	PORTAGE	PALMYRA TWP	KALE CREEK	CRUDE OIL	2160	GAL	0
89	W C I STEEL	TRUMBULL	WARREN	MAHONING RIV ER	WASREWATER	18000	GAL	0
89	W C I STEEL	TRUMBULL	WARREN	MAHONING RIVER	COOLING WATER	0	UNK	0
89	W C I STEEL	TRUMBULL	WARREN	MAHONING RIVER	WASTEWATER	0	UNK	0
89	W C I STEEL	TRUMBULL	WARREN	MAHONING RIVER	WASTE WATER	2000	GAL	0
90	W C I STEEL	TRUMBULL	WARREN	MAHONING RIVER	OIL	0	UNK	0
91	W C I STEEL	TRUMBULL	WARREN	MAHONING RIVER	LUBE OIL	30	GAL	0
92	W C I STEEL	TRUMBULL	WARREN	MAHONING RIVER	HYDROCHLORIC ACID	1600	GAL	0
92	W C I STEEL	TRUMBULL	WARREN	MAHONING RIVER	WASTE WATER	0	UNK	0
92	W C I STEEL	TRUMBULL	WARREN	MAHONING RIVER	WASTE WATER	12000	GAL	0
92	W C I STEEL	TRUMBULL	WARREN	MAHONING RIVER	WASTE WATER	0	UNK	0
92	W C I STEEL	TRUMBULL	WARREN	MAHONING RIVER	OIL	0	UNK	0
92	W C I STEEL	TRUMBULL	WARREN	MAHONING RIVER	WASTE WATER	0	UNK	0
93	W C I STEEL	TRUMBULL	WARREN	MAHONING RIVER	OIL	0	UNK	0
92	W C I STEEL	TRUMBULL	WARREN	MAHONING RIVER	WASTE WATER	0	UNK	0
92	W C I STEEL	TRUMBULL	WARREN	MAHONING RIVER	STORM WATER	0	UNK	0
92	W C I STEEL	TRUMBULL	WARREN	MAHONING RIVER	WASTE WATER	0	UNK	0
92	W C I STEEL	TRUMBULL	WARREN	MAHONING RIVER	WASTE WATER	100	GAL	0
92	W C I STEEL	TRUMBULL	WARREN	MAHONING RIVER	WASTE WATER	2000	GAL	11000
92	W C I STEEL	TRUMBULL	WARREN	MAHONING RIVER	HYDRAULIC OIL	0	UNK	0
92	W C I STEEL	TRUMBULL	WARREN	MAHONING RIVER	UNTREATED RECYCLED W	0	UNK	0
93	W W OPERATION SERVICES	MAHONING	BOYD	MAHONING RIVER	SUSPENDED SOLIDS	0	UNK	0
89	WARREN STP	TRUMBULL	WARREN	MAHONING RIVER	SEWAGE	0	UNK	0
93	WARREN STP	TRUMBULL	BAZETTA TWP	MOSQUITO CREEK	ALUM SLUDGE	150000	GAL	0
91	WARREN WATER PLANT	TRUMBULL	WARREN	MOSQUITO CREEK TRIB	POTASSIUM PERMANGANA	0	UNK	0
89	WEAN IND	MAHONING	YOUNGSTOWN	BEARS DEN RUN	OIL	0	UNK	0
90	WEIMER ENTERPRISES	MAHONING	BERLINE CENTER	UNKNOWN	GASOLINE	18	GAL	0
90	WESTERN RESERVE FARM CO	PORTAGE	RAVANNA	UNKNOWN	28% LIQUID NITROGEN	11000	GAL	0
89	WILLOW CREEK LANDFILL	PORTAGE	ATWATER	BERLIN RESERVOIR	LEACHATE	0	UNK	0
94	WINDHAM MOBIL	PORTAGE	WINDHAM	UNKNOWN CREEK	KEROSENE	100	GAL	0
93	WINDHAM MOBIL SERVICE	PORTAGE	WINDHAM	EAGLE CREEK	GASOLINE	0	UNK	0
90	WINDHAM STP	PORTAGE	WINDHAM	SF EAGLE CREEK	SEWAGE	30000	GAL	0
94	WINDHAM STP	PORTAGE	WINDHAM	SF EAGLE CREEK	SEWAGE	0	UNK	0
89	WISE OIL CO	PORTAGE	RAVENNA	STORM SEWER	GASOLINE	0	UNK	0
90	YELLOW FREIGHT	TRUMBULL	LIBERTY	UNKNOWN	DIESEL FUEL	40	GAL	0
91	YELLOW FREIGHT SYSTEMS	MAHONING	CANFIELD TWP	UNKNOWN	DIESEL FUEL	50	GAL	0
89	YOUNGSTOWN STP	MAHONING	YOUNGSTOWN	MAHONING RIVER	SEWAGE	0	UNK	0
91	YOUNGSTOWN STREET DEPT	MAHONING	YOUNGSTOWN	STORM SEWER	PAINT	0	UNK	0
91	YOUNGSTOWN WELDING & EN	MAHONING	AUSTINTOWN TW	FOUR MILE RUN	NITRIC ACID	200	GAL	0
91	YOUNGSTOWN WELDING & EN	MAHONING	AUSTINTOWN TW	STORM SEWER-BEARS DEN RU	HYDRAULIC OIL	50	GAL	30
93	YOUNGSTOWN WELDING & EN	MAHONING	YOUNGSTOWN	STORM SEWER	HYDROFLURIC ACID	106	LBS	0
92	YOUNGSTOWN WELDING & EN	MAHONING	YOUNGSTOWN	FOUR MILE RUN	OIL	0	UNK	0

APPENDIX 4

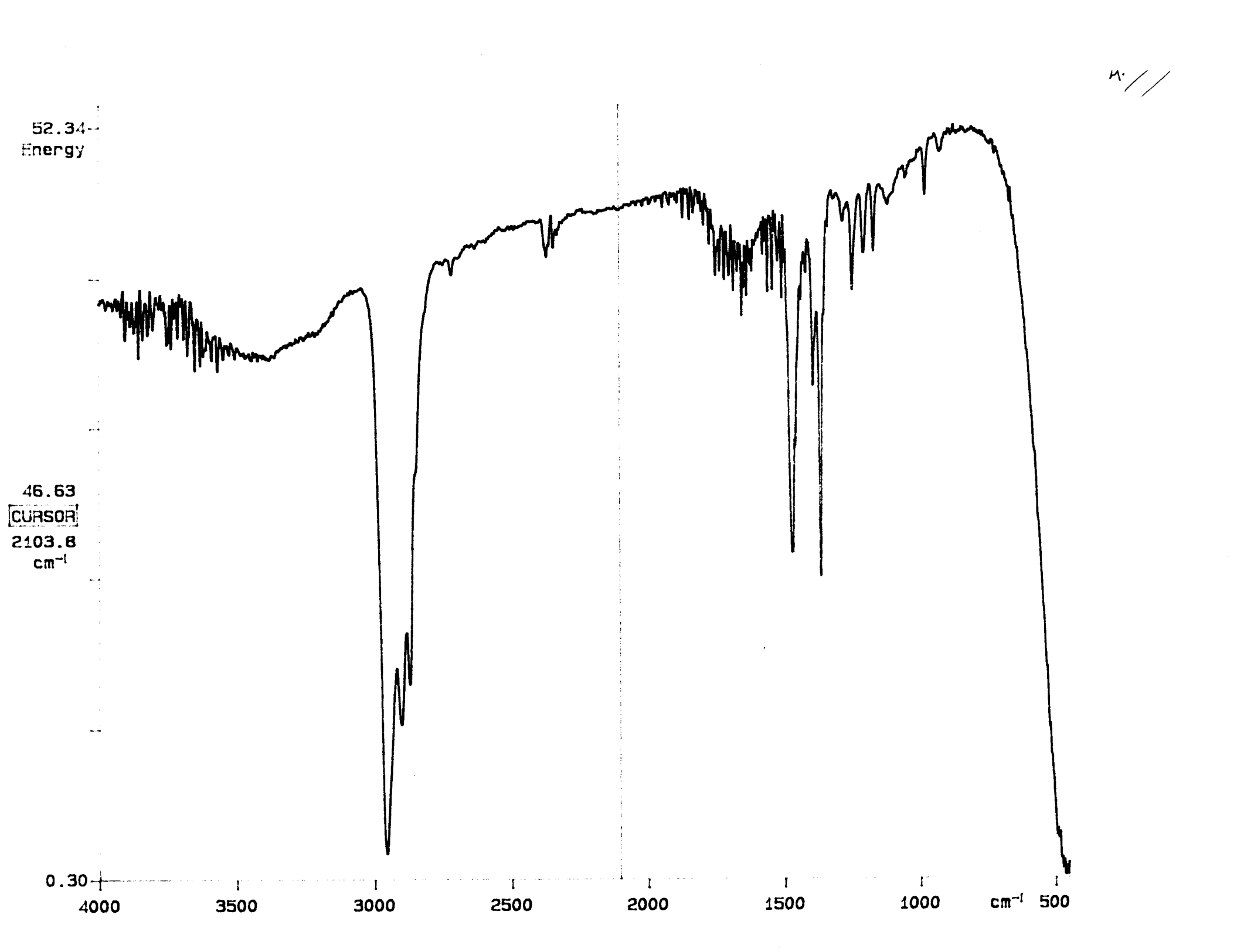
IR SPECTRA

First Soil Sample-taken from the patient's yard
Collected 10.22.98
Sample run 4.29.99

This sample was stored, after collection, in cleaned aluminum foil within a labeled plastic bag at 4°C until further work-up was possible.

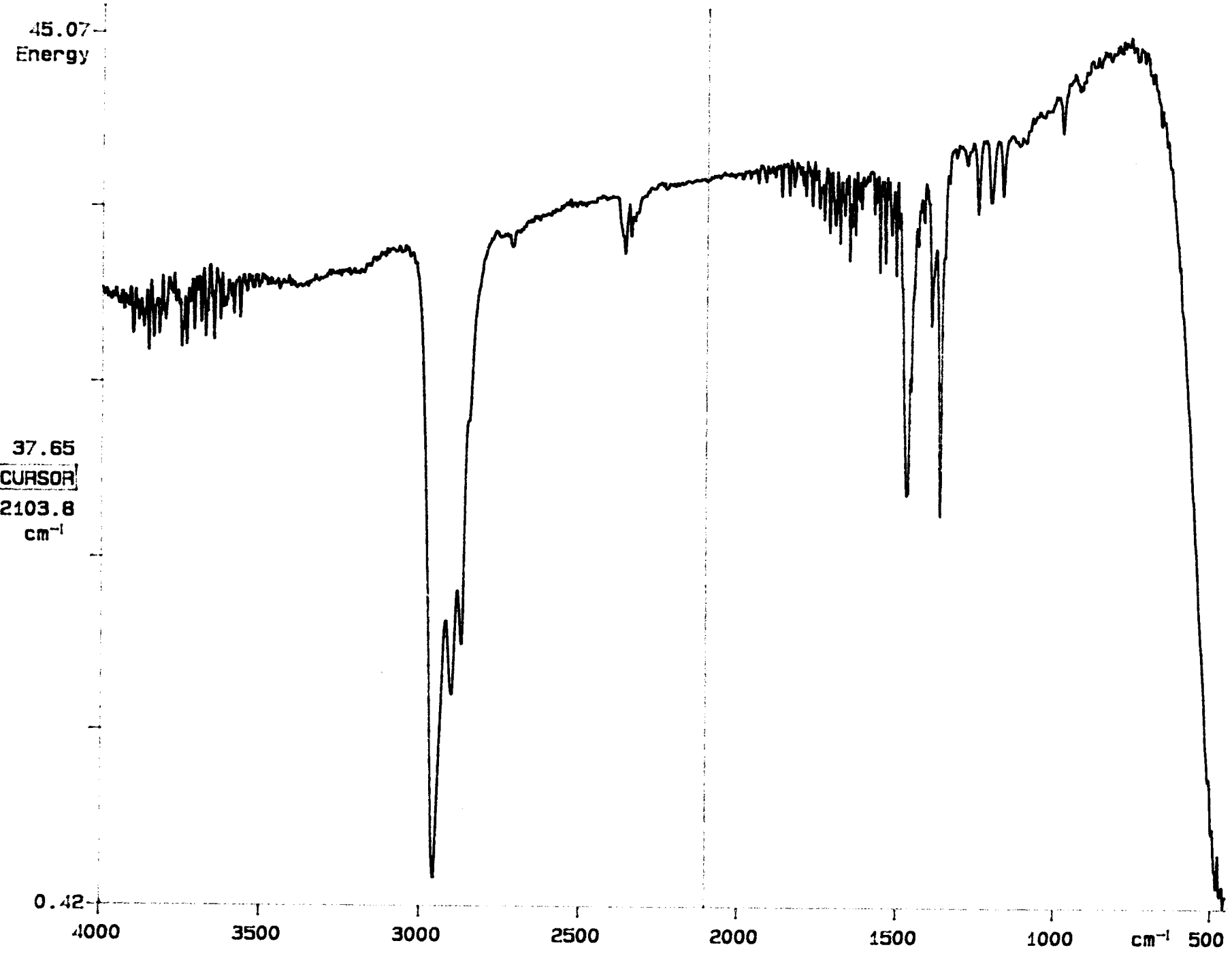
The soil was later thawed and manually mixed to promote homogeneity. Approximately 15.271g of soil was mixed with sodium sulfate to remove any water. Th dried soil was spiked with 452ng PCB-103, transferred to a clean cellulose thimble and extracted via soxhlet with dichloromethane, 24h. The extract was then reduced via rotary evaporation, transferred into hexanes and concentrated under nitrogen, to 2ml.

The extract was then cleaned via an alumina column composed of a glass wool plug, on top of which was 2g Al_2O_3 and 1cm Na_2SO_4. The alumina column was pre-prepared with 5ml of 5% dichloromethane in petroleum ether. Th resulting eluent was then concentrated and solvent exchanged into iso-octane under nitrogen.


52.34
Energy
46.63
CURSOR
2103.8
cm-1
0.30
4000
3500
3000
2500
2000
1500
1000
cm-1
500

Second Soil Sample-taken from patient's yard
Collected 10.22.98
Sample run 4.30.99

This sample was prepared in accordance with the protocol as outlined for the first soil sample.


45.07
Energy
37.65
CURSOR
2103.8
cm⁻¹
0.42
4000
3500
3000
2500
2000
1500
1000
cm⁻¹
500

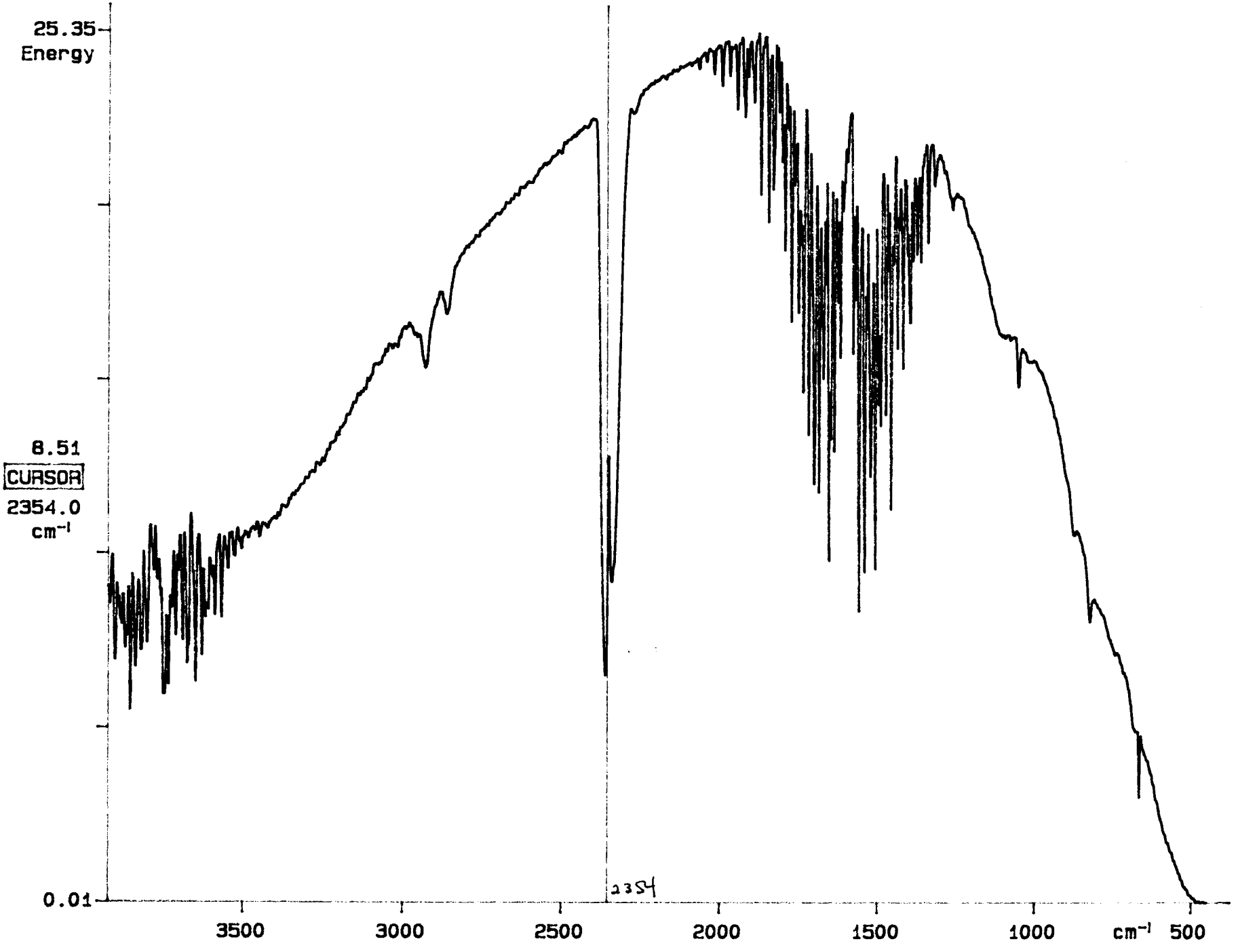
Water sample-(filter) 3B Feed
3B Feed-area corresponds to the map of Berlin Lake, located in Appendix 3
Collected 10.22.98
Sample run 5.3.99

The water samples were collected in cleaned 4L solvent jugs, from locations in and around Berlin Lake. The samples were stored at 4°C until extraction was possible.

The polyurethane foam plugs were extracted via soxhlet, in petroleum ether, for 24h. The filters, prior to use, were refluxed in dichloromethane for 18h.

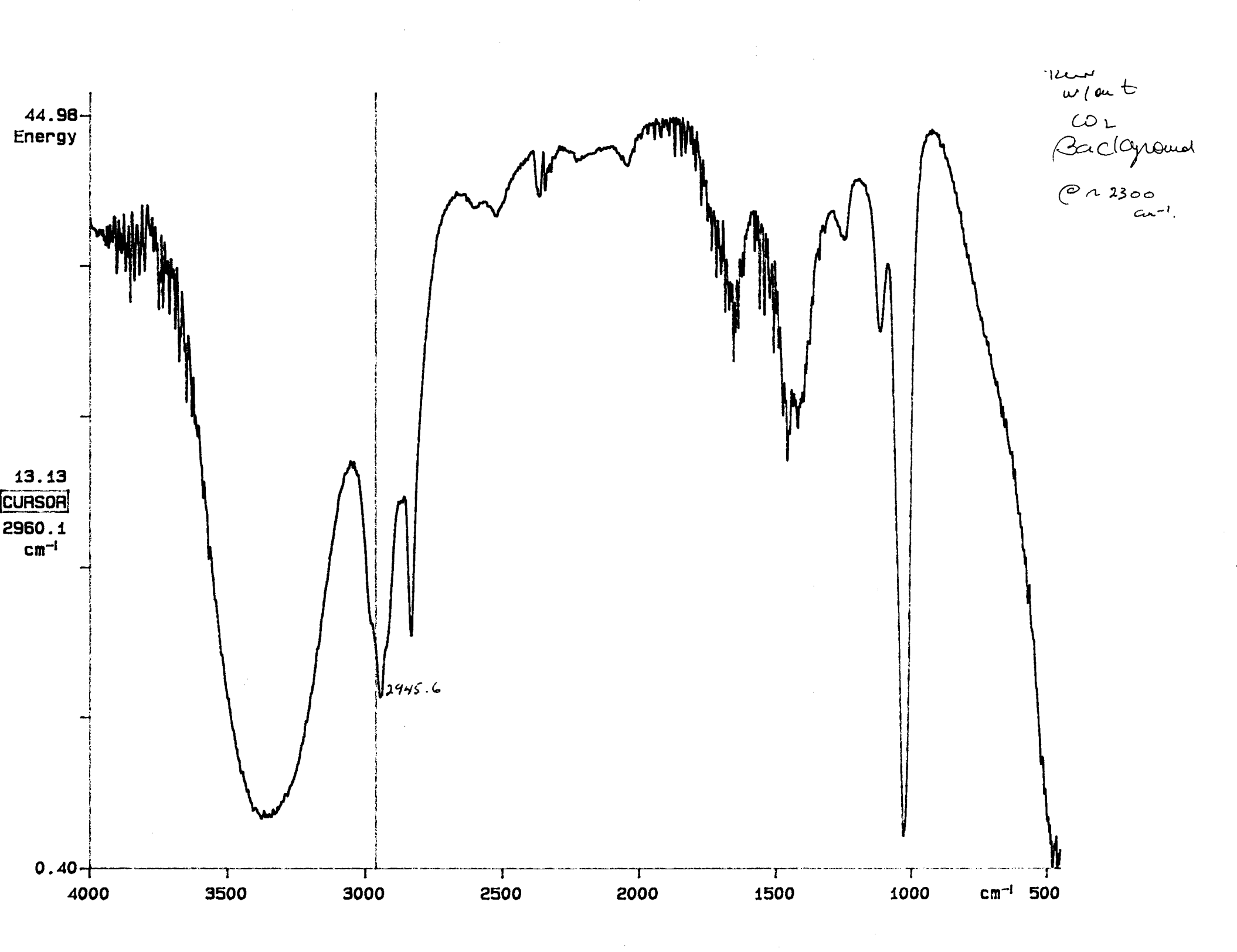
The water samples were transferred into individual stainless steel canisters. The water samples were pushed, via nitrogen pressure, through a 47mm GMF water filter, in attempt to remove any particulate matter; each sample required several filters, due to high levels of particulate matter. The water filters were then wrapped individually in cleaned aluminum foil and stored in plastic bags at -10°C.

The filters were soxhlet extracted with dichloromethane 24h. The extracts were then reduced to 5-10ml and solvent exchanged into iso-octane via rotary evaporation. The entire sample inventory was reduced individually to 1ml under nitrogen. The samples were cleaned using a silicic acid/alumina column; a glass column was dry-packed with a first layer of 3g silicic acid)1.7% water added), followed by a second layer of 2g adsorption alumina (6% water added), and a third layer of 2cm anhydrous sodium sulfate.


25.35
Energy
8.51
CURSOR
2354.0
cm⁻¹
0.01
2354
3500
3000
2500
2000
1500
1000
cm⁻¹
500

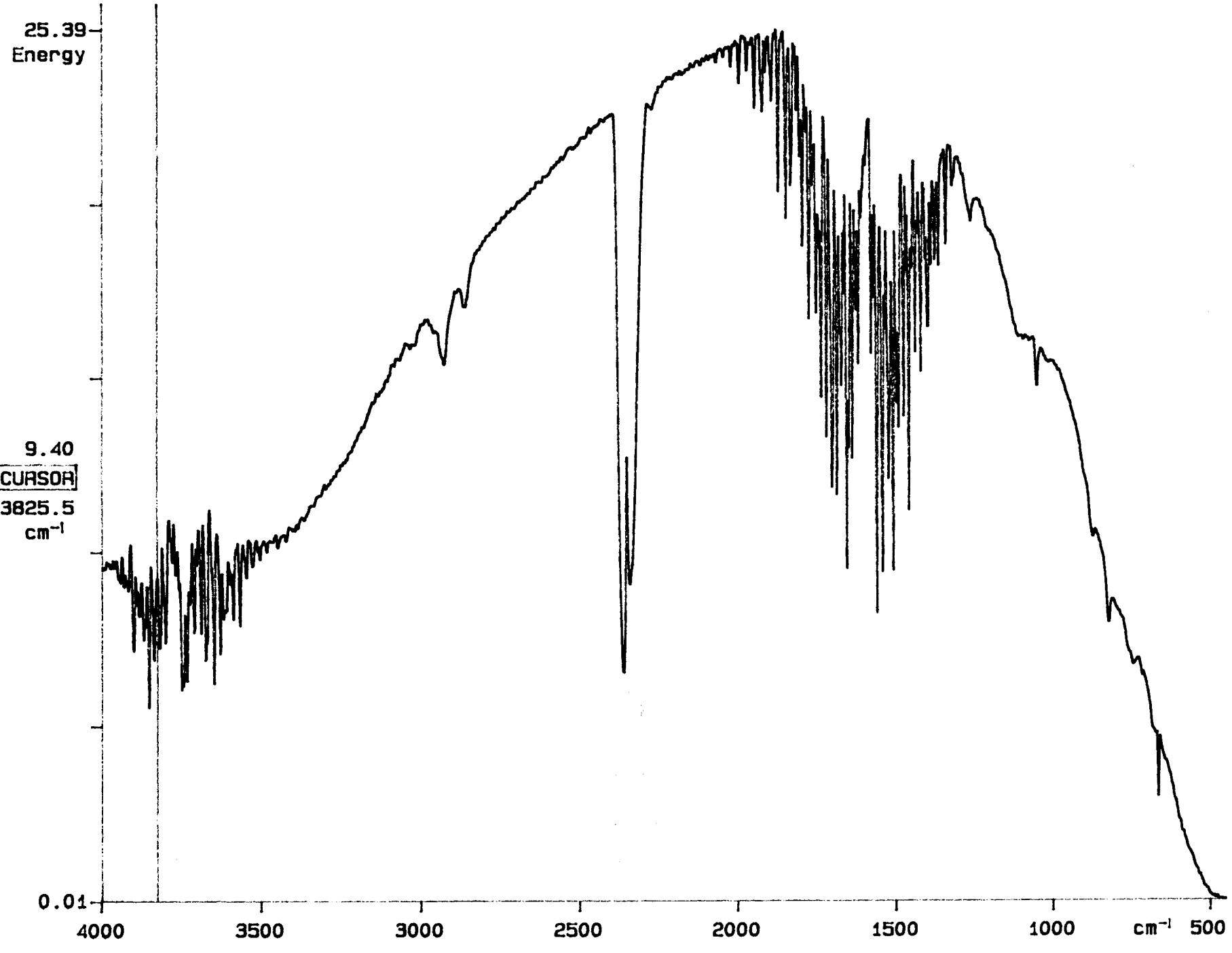
Water sample-(syringe) B Feed
B Feed-area corresponds to the map of Berlin Lake, located in
Appendix 3
Collected 10.22.98
Sample run 5.3.99

This sample was prepared in accordance with the protocol as outlined for
The first water sample.


44.98
Energy
13.13
CURSOR
2960.1
cm-1
0.40
4000
3500
3000
2500
2000
1500
1000
cm-1
500
2945.6
New w/out CO2 Background @ ~2300 cm-1.

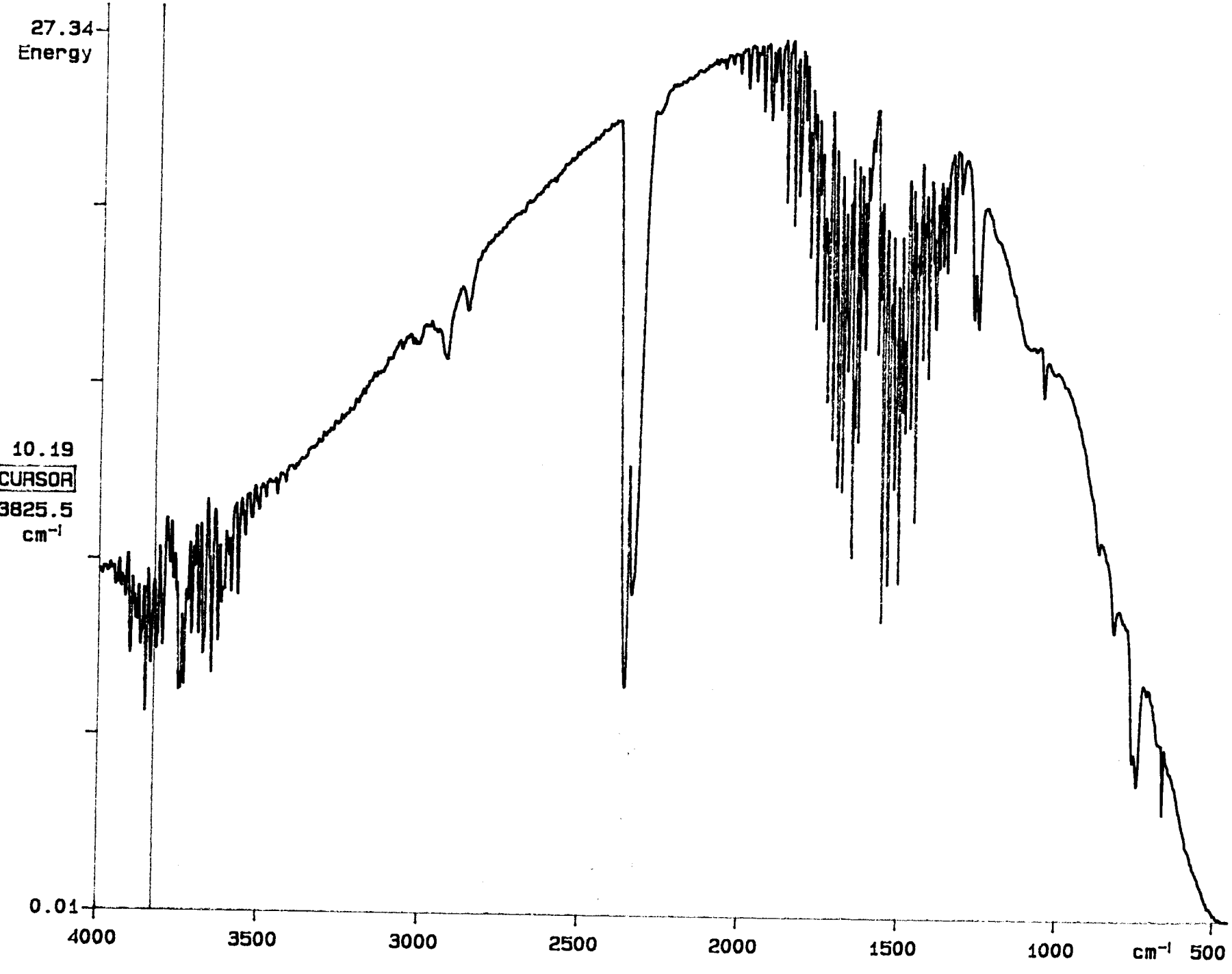
Water sample-(filter) 4B Feed
4B Feed-area corresponds to the map of Berlin Lake, located in
Appendix 3
Collected 10.22.98
Sample run 5.2.99

This sample was prepared in accordance with the protocol as outlined for
The first water sample.


25.39
Energy
9.40
CURSOR
3825.5
cm-1
0.01
4000
3500
3000
2500
2000
1500
1000
cm-1
500

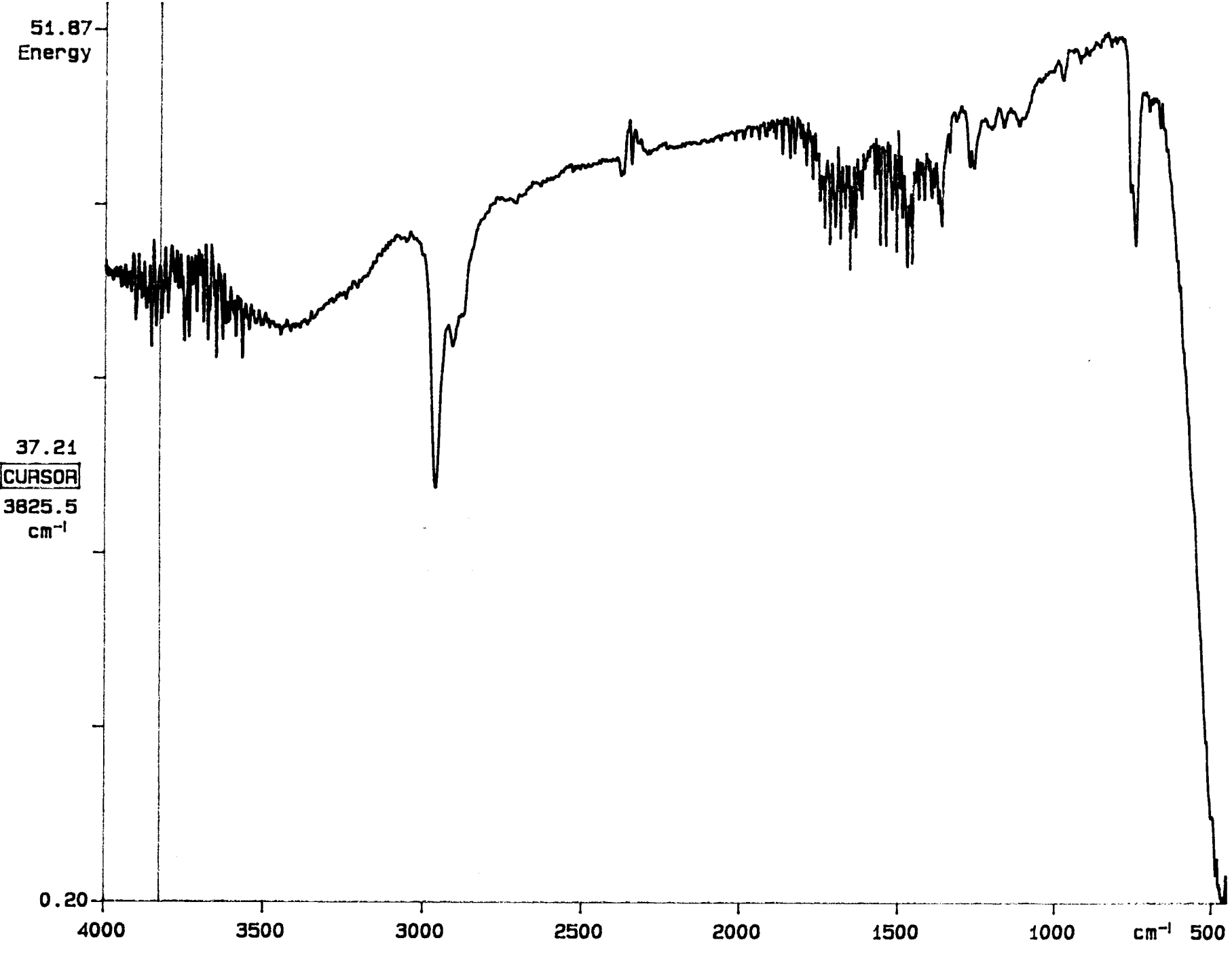
Water sample-(filter) 2B Feed
2B Feed-area corresponds to the map of Berlin Lake, located in
Appendix 3
Collected 10.22.98
Sample run 5.3.99

This sample was prepared in accordance with the protocol as outlined for
The first water sample.


27.34
Energy
10.19
CURSOR
3825.5
cm-1
0.01
4000
3500
3000
2500
2000
1500
1000
cm-1
500

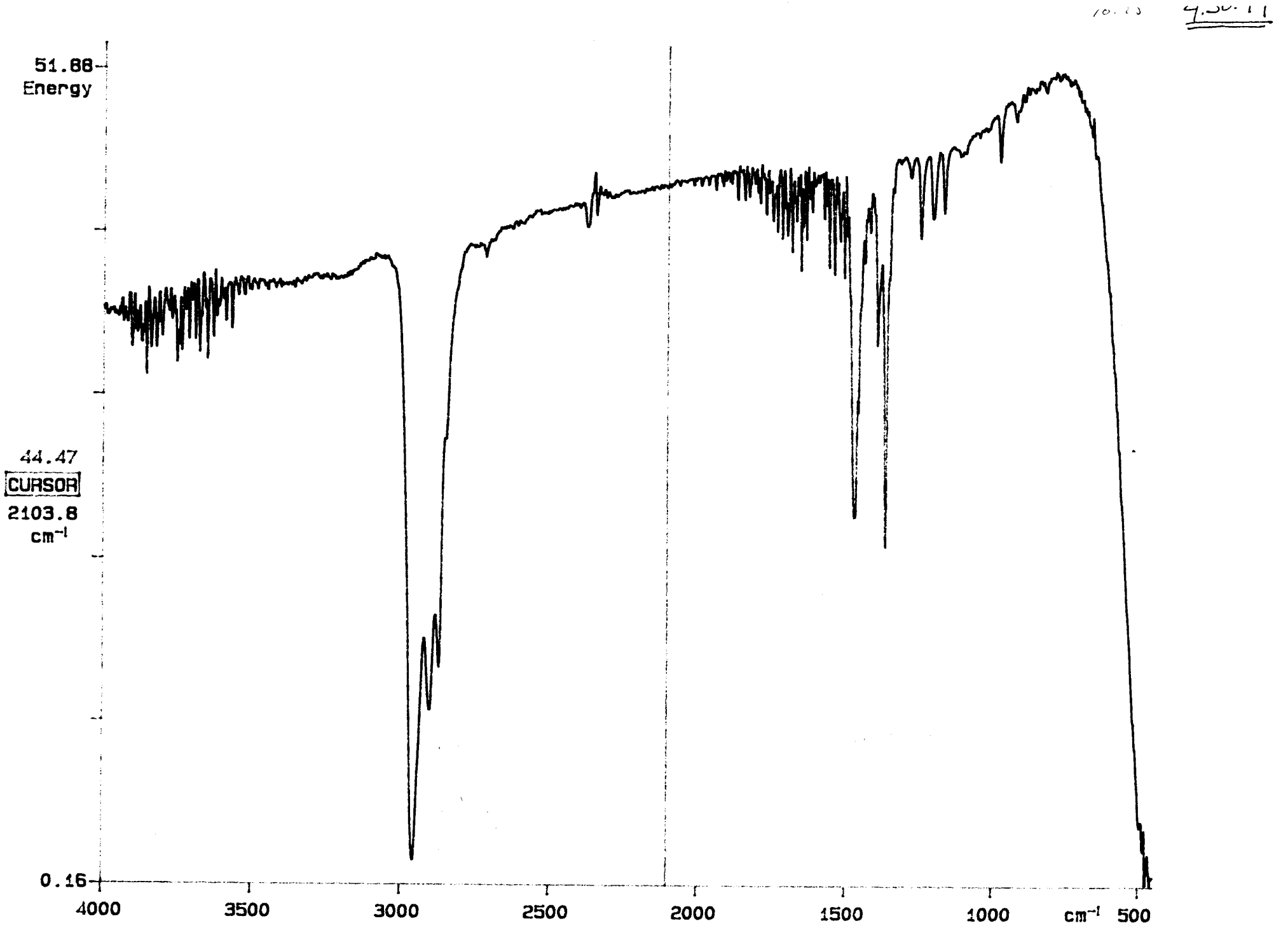
Water sample-(filter) 1B Feed
1B Feed-area corresponds to the map of Berlin Lake, located in Appendix 3
Collected 10.22.98
Sample run 5.2.99

This sample was prepared in accordance with the protocol as outlined for the first water sample.


51.87
Energy
37.21
CURSOR
3825.5
cm⁻¹
0.20
4000
3500
3000
2500
2000
1500
1000
cm⁻¹
500

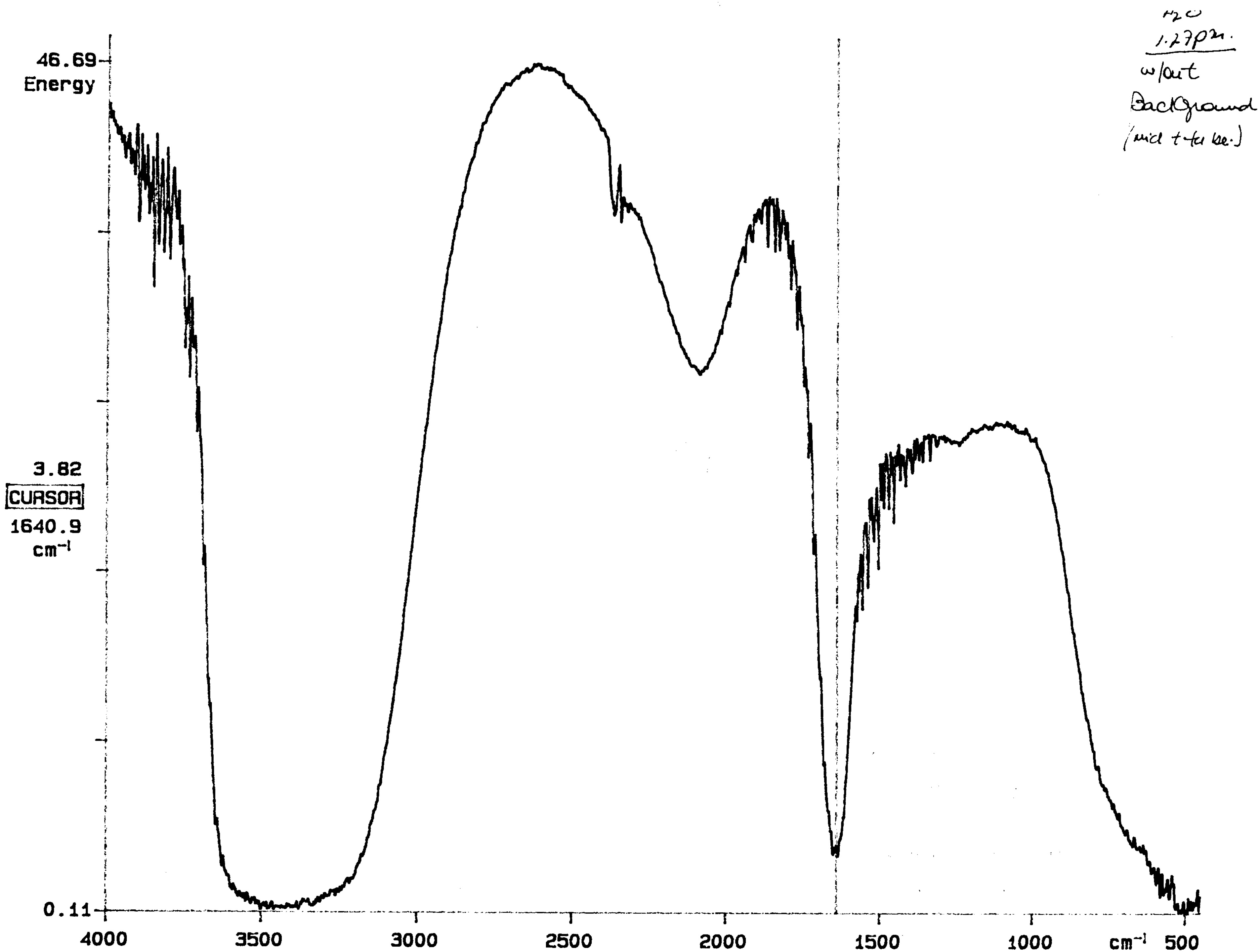
Water sample·(filter) C Feed
C Feed·area corresponds to the map of Berlin Lake, located in
Appendix 3
Collected 10.25.98
Sample run 4.30.99

This sample was prepared in accordance with the protocol as outlined for the first water sample.


51.88
Energy
44.47
CURSOR
2103.8
cm⁻¹
0.16
4000
3500
3000
2500
2000
1500
1000
cm⁻¹
500

Water sample-(filter) 1B Feed
1B Feed-area corresponds to the map of Berlin Lake, located in Appendix 3
Collected 10.25.98
Sample run 5.4.99

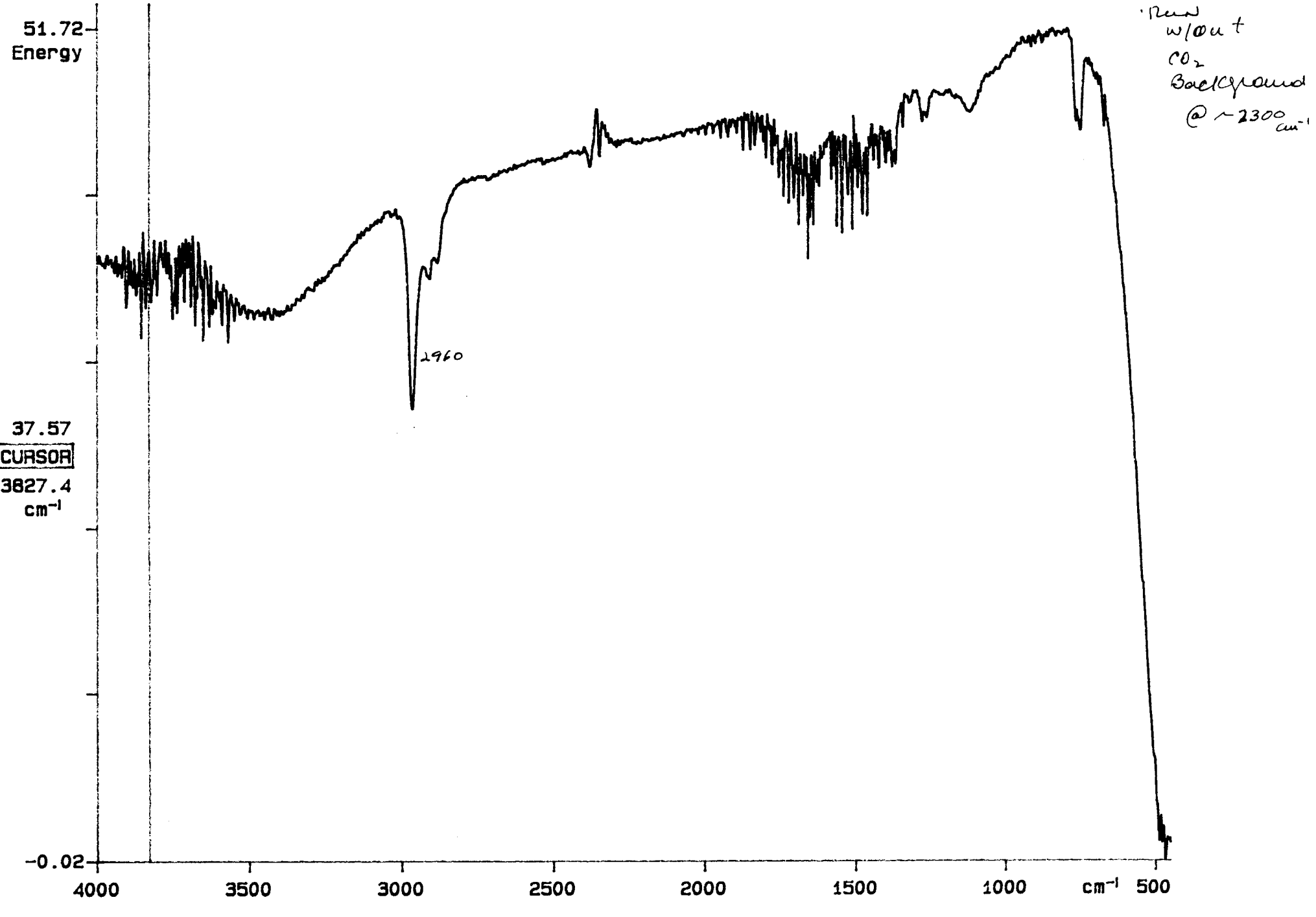
This sample was prepared in accordance with the protocol as outlined for the first water sample.



46.69
Energy
3.82
CURSOR
1640.9
cm⁻¹
0.11
4000
3500
3000
2500
2000
1500
1000
cm⁻¹ 500
H2O
1.27pm.
w/out
Background.
(mid + far bee.)

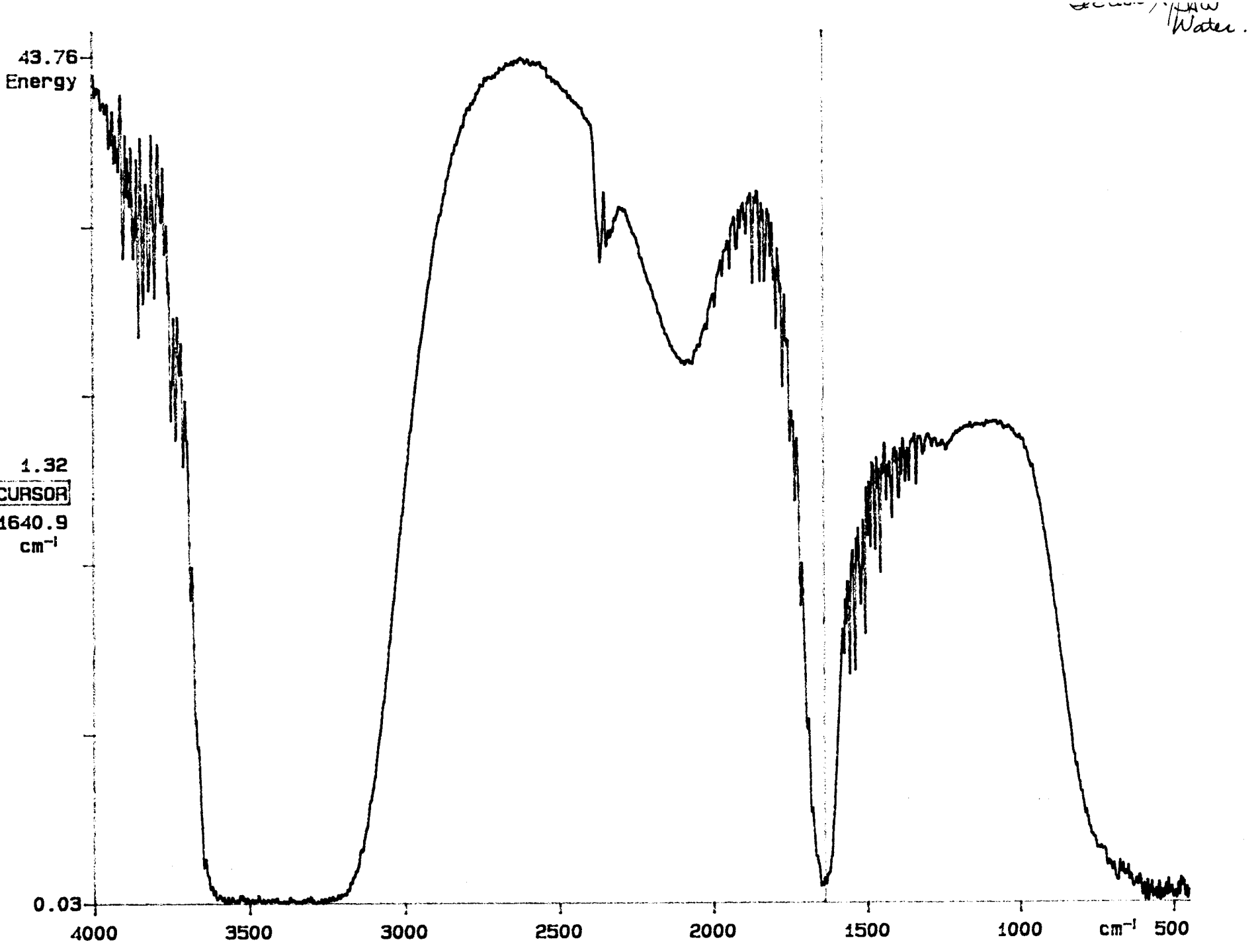
Water sample-(filter) 2B Feed
2B Feed-area corresponds to the map of Berlin Lake, located in Appendix 3
Collected 10.25.98
Sample run 5.3.99

This sample was prepared in accordance with the protocol as outlined for the first water sample.


51.72
Energy
37.57
CURSOR
3827.4
cm⁻¹
-0.02
1960
4000
3500
3000
2500
2000
1500
1000
cm⁻¹
500
Run w/out CO₂ Background @ ~2300 cm⁻¹

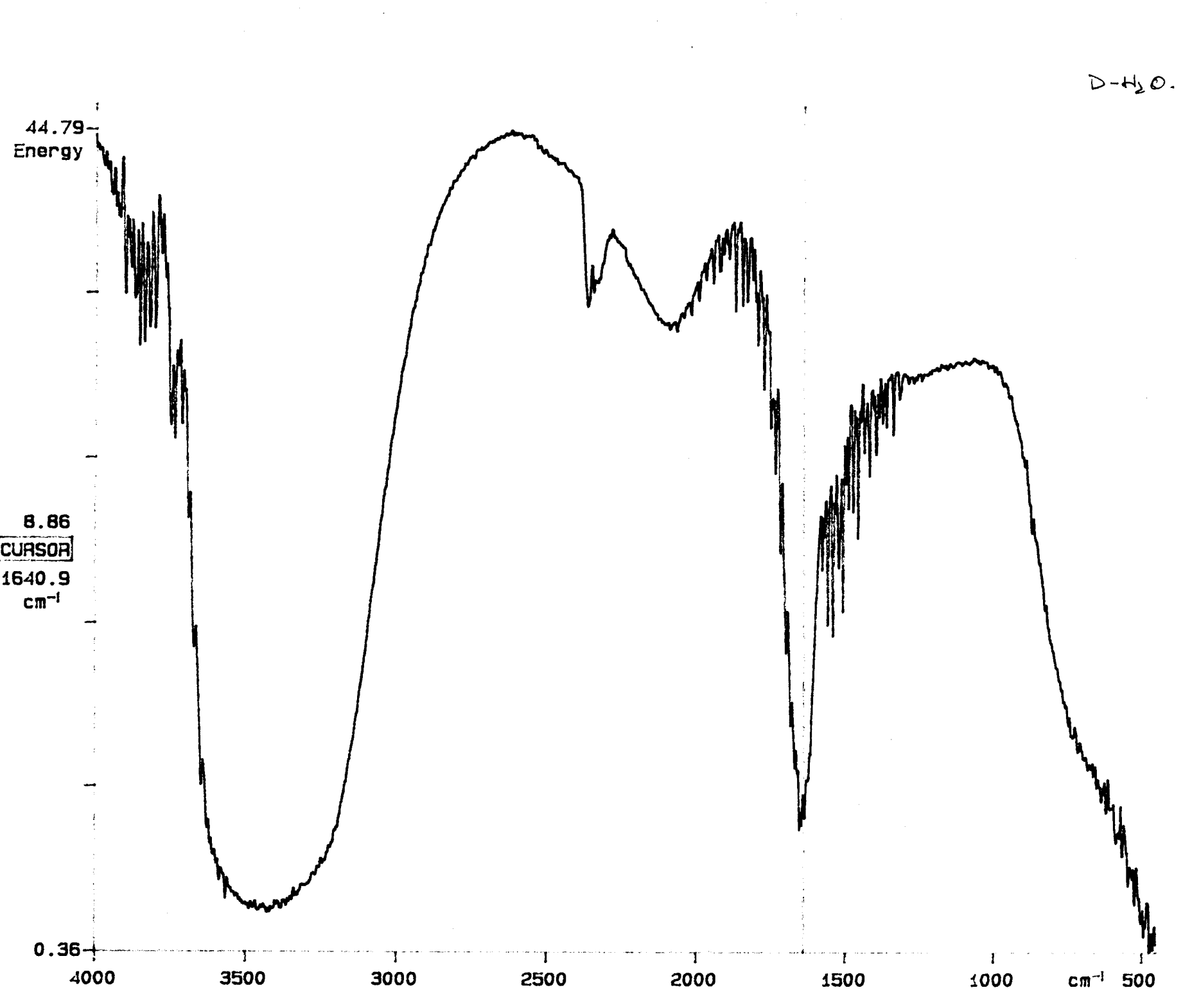
Water sample-(filter) Section A/Dam Water
Section A/Dam Water-area corresponds to the map of Berlin Lake, located in Appendix 3
Collected 10.25.98
Sample run 5.18.99

This sample was prepared in accordance with the protocol as outlined for the first water sample.


Water.
43.76
Energy
1.32
CURSOR
1640.9
cm⁻¹
0.03
4000
3500
3000
2500
2000
1500
1000
cm⁻¹
500

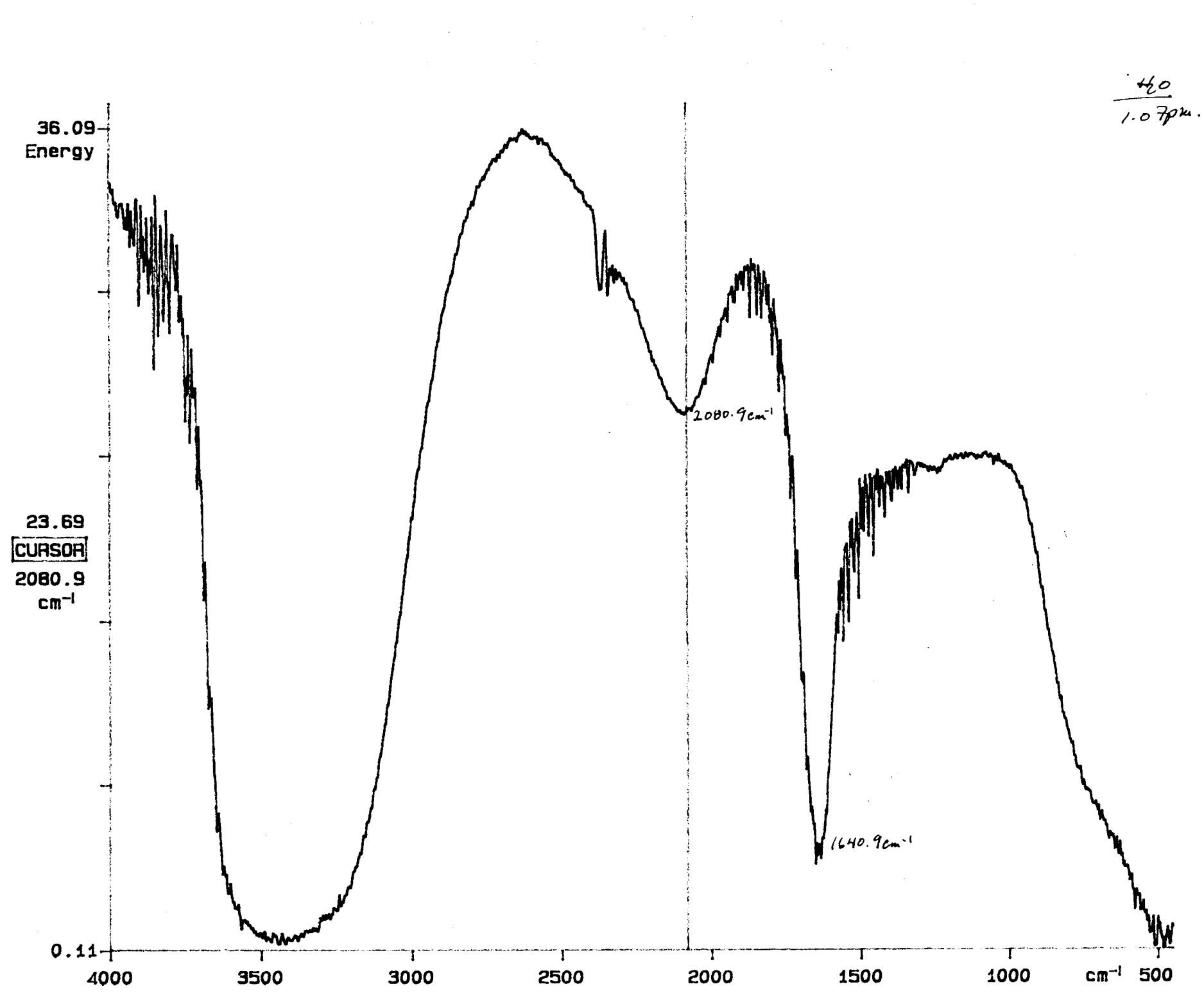
Water sample-(filter) Dam Water
Dam Water-area corresponds to the map of Berlin Lake, located in Appendix 3
Collected 10.25.98
Sample run 5.18.99

This sample was prepared in accordance with the protocol as outlined for the first water sample.


D-H2O.
44.79
Energy
8.86
CURSOR
1640.9
cm-1
0.36
4000
3500
3000
2500
2000
1500
1000
cm-1
500

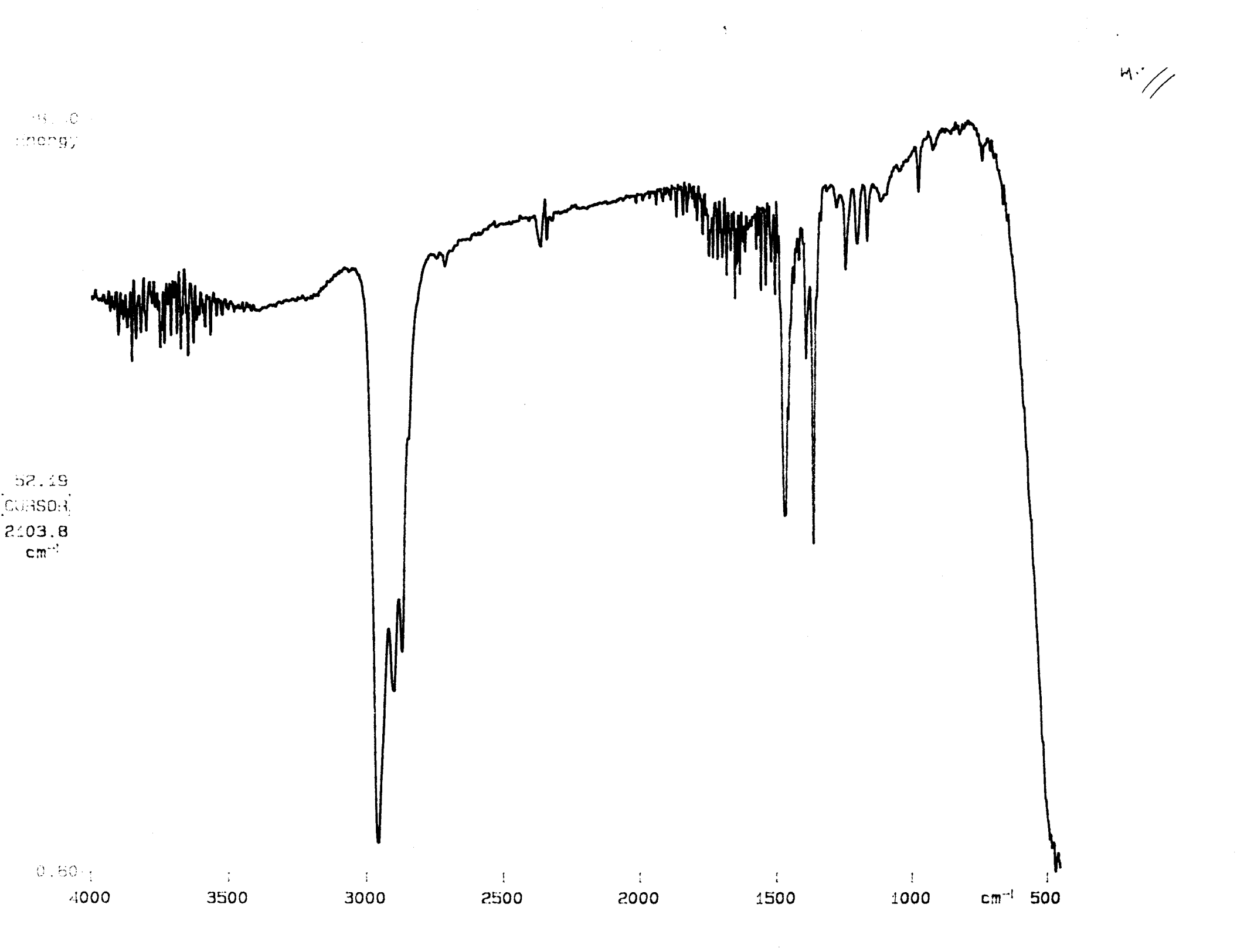
Water sample-(filter) Beyond Dam
Beyond Dam-area corresponds to the map of Berlin Lake, located
In Appendix 3
Collected 10.25.98
Sample run 5.4.99

This sample was prepared in accordance with the protocol as outlined for the first water sample.


40
1.07pm.
36.09
Energy
23.69
CURSOR
2080.9
cm-1
0.11
2080.9cm-1
1640.9cm-1
4000
3500
3000
2500
2000
1500
1000
cm-1
500

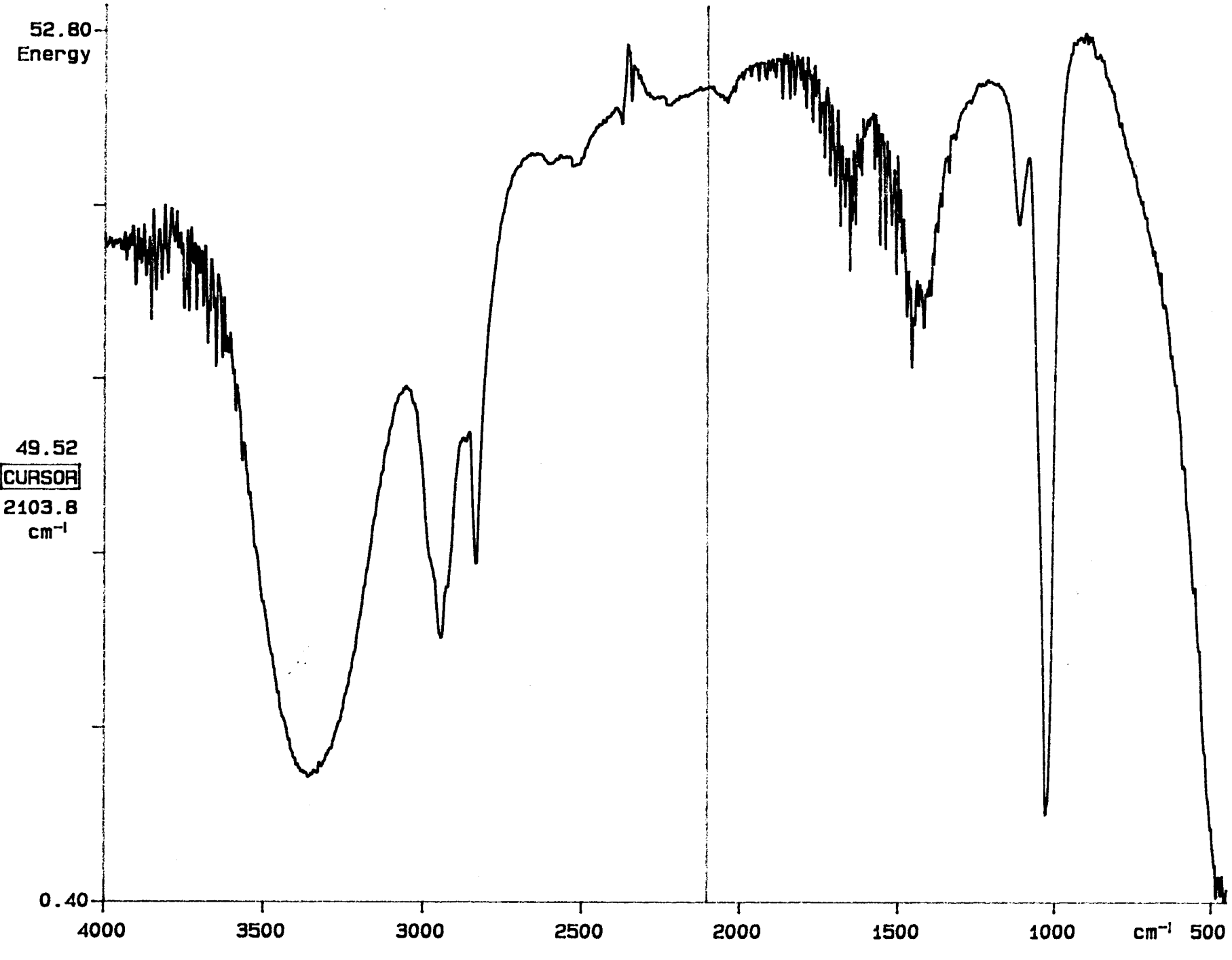
Water sample-(filter) C Feed
C Feed-area corresponds to the map of Berlin Lake, located
In Appendix 3
Collected 10.25.98
Sample run 4.29.99

This sample was prepared in accordance with the protocol as outlined for the first water sample.


52.19
CURSOR
2103.8
cm-1
0.60
4000
3500
3000
2500
2000
1500
1000
cm-1
500

Water sample-(syringe) C Feed
C Feed-area corresponds to the map of Berlin Lake, located
In Appendix 3
Collected 10.25.98
Sample run 5.3.99

This sample was prepared in accordance with the protocol as outlined for the first water sample.


52.80
Energy
49.52
CURSOR
2103.8
cm-1
0.40
4000
3500
3000
2500
2000
1500
1000
cm-1
500

APPENDIX 5
HUMAN SUBJECTS PROTOCOL REVIEW FORM

Youngstown State University / One University Plaza / Youngstown, Ohio 44555-0001

May 31, 2000

Dr. Peter J. Kasvinsky
Dean
School of Graduate Studies
Youngstown State University
CAMPUS

Dear Dean Kasvinsky:

This is to report on the results of the administrative review of human subjects activity related to the thesis proposal of Ms. Meredith Tuttle, M.S. candidate in Chemistry, entitled "The Convergence of Environmental Influences as Potential Precipitating Factors of AML-M2," which was prepared under the advisement of Dr. Daryl Mincey, Chairman, Department of Chemistry.

Although the research was not federally-funded, and consequently not materially subject to federal regulations, as you directed, a rigorous review of the human subjects-related aspects of Ms. Tuttle's thesis research was nonetheless conducted using expedited protocol procedures consistent with U.S. Department of Health & Human Services, National Institutes of Health, Office of Extramural Research, Office for Protection from Research Risks guidelines. The reviewers consisted of: YSU Human Subjects Research Committee (HSRC) Program Chairperson, JoLynn Carney, Ph.D., Anita Hakstedde, M.D., who served as expert biomedical reviewer, and Eric C. Lewandowski, Certified Research Administrator, in his capacity as HSRC Administrative Co-chair.

Each of the reviewers was provided a copy of the full committee human subjects protocol form, prepared by Ms. Tuttle, as well as access to the full thesis under consideration. As is customary in expedited protocol reviews, this review was conducted via correspondence.

Based on the review, the consensus findings were, and are, that: (1) the nature of the study, being essentially a review of medical records, allowed no subject harm, and reflects activity that normally qualifies for exemption from full committee review under DHHS Category 4 exemption; (2) the study utilized data that was voluntarily provided to the investigator by persons authorized to release it; (3) the investigator, acting in a good faith manner, provided background information to the purveyors of the subject data sufficient for them to form an adequate judgment with respect to the elements of informed consent, and to allow its release without coercion; (4) the investigator properly utilized the data collected for the purpose of her thesis development and exercised mature academic consideration and discretion in its use.

um, the reviewers commend the intuitively sensitive approach to human subject data
ɔction and use employed by Ms. Tuttle in conducting her study. At the same time,
reviewers earnestly hope that thesis committee members, in future, will appreciate
weight of their responsibility to correctly inform candidates of proper IRB practice,
will adhere to the institutionally-defined IRB process.

ɔectfully submitted,

Lynn Carney (EC)

ynn Carney, Ph.D.
ɡram Chairperson

Eric C. Lewandowski, CRA
Administrative Co-chair

/ECL

Dr. Darryl Mincey
Ms. Meredith Tuttle

e Received _____
e completed by Human Subjects Secretary)

Protocol # _____
To be completed by Human Subjects Secretary)

YOUNGSTOWN STATE UNIVERSITY
Human Subjects Protocol Review Form

ıcipal Investigator* Daryl Mincey, Ph.D. | Chemistry-Chair 742-3663
student investigators, advisor's name first) — Typed Name & Title | Department Name & Telephone #

Investigator* Meredith Tuttle,Masters Candidate | Chemistry 783-3224
Typed Name & Title | Department Name & Telephone #

Investigator* _____ | _____
Typed Name & Title | Department Name & Telephone #

ıse Note: Do not list collaborators from other institutions here unless they hold approved joint appointment(s) at YSU

e of Study The Convergence of Environmental Influences as Potential Precipitating Factors of AML-M2

xternal Funding Involved? ☒ NO ☐ YES
se check appropriate box)

es, Type in Name of Funding Agency/Program _____

tivity Start Date _____ End Date _____ Anticipated Funding Date _____

Collaborating Institutions Involved? ☒ NO ☐ YES
se check appropriate box)

es, Type in the Following _____ (Institution Name) _____ (Name & Title of Chief Collaborator)

_____ (Institution Name) _____ (Name & Title of Chief Collaborator)

ıis Study Subject to Other itutional Human Subjects Review? ☒ NO ☐ YES

es, Type in the Following _____ (Institution Name) _____ (Protocol Review Date/Determination)

itution with Primary Review Responsibility ☒ YSU ☐ OTHER (Please Identify) _____

INSTRUCTIONS TO INVESTIGATORS

purpose of an institutional human subjects review is to foster academic inquiry through the study of ıan processes and behavior, while protecting subject rights and interests. The following questions are nded to promote both of these ends. Please answer each question below accurately, completely and anguage comprehensible to an informed layperson. Attach additional pages as necessary. Requests for her information or clarification of issues or questions related to human subjects research or this protocol ı be directed to the current co-chairs of the YSU Human Subjects Committee via the Office of Grants and nsored Programs (Telephone 742-2377). Please type all responses on this form and any attachments.

Briefly describe the nature of the activity you are proposing to conduct involving human subjects.
Please try to limit your response to the space provided, and be sure to address the following: (A) the purpose of the research and the hypotheses to be tested; (B) short references to the pertinent scientific literature; (C) an overview of the research design, method and mode of analysis; (D) an appraisal of the anticipated value of the research to the investigator(s), the human subjects, YSU, the scientific community, and society-at-large; (E) the specific site(s) of the research; and (F) investigator access to them.

- The purpose of this research is to explore the potential environmental factors contributing to acute myelogenous leukemia-M2; working upon the hypothesis that a convergence of environmental influences may influence the development of this leukemia.

- medical texts; scientific journals; medical journals; pharmaceutical manufacturer product inserts; extant medical records

- collection of soil and water samples with lab work-up and analysis; literature search with extant medical record corroboration

- to further elucidate the potential environmental influences on acute myelogenous leukemia

- YSU labs

- graduate student at YSU

Please describe the target population in specific terms. Be sure to provide detail about numbers of subjects, age, gender, physical condition or any other information that establishes the parameters of the population of your study.

lv 10 year old cousin diagnosed with acute myelogenous leukemia-M2 (male)

Briefly describe each of the different conditions or manipulations to be conducted in the study.

:eview of cousin's extant medical records

Briefly describe the nature of the measures or observations that will be taken in the study.

:view of cousin's extant medical records

If any questionnaires, tests, or other instruments are to be used, please provide a brief description and either a copy or an indication of when a copy will be submitted to the Committee for review.

N/A

Will the subjects encounter the possibility of psychological, social, physical or legal risk, that is, the probability of harm or injury occurring as a result of participation in this research study?
☐ Yes ☒ No **If so, please describe.**

Will the study involve any stress, that is, any physical, chemical or emotional factors that may cause bodily or mental tension and may be a factor in causing disease? ☐ Yes ☒ No
If so, please describe.

Will the subjects be deceived or misled in any way? ☐ Yes ☒ No
If so, please describe and include a statement regarding the nature of their debriefing.

Will there be any probing for information that an individual might consider to be personal or sensitive? ☐ Yes ☒ No **If so, please describe.**

Will subjects be presented with materials that they might regard to be offensive, threatening, or degrading? ☐ Yes ☒ No **If so, please describe.**

Approximately how much time will be required of each subject?

No time commitment

How will subjects for this study be solicited or contacted?

My help was solicited by my family

What steps will be taken to insure that subjects' participation is voluntary? What inducements will be offered to subjects for their participation? What is the source of those inducements?

Please refer to #12

It is important that subjects be informed regarding the general nature of the proposed human subject activity, especially including a description of anything they may consider unpleasant or risky. Please provide a statement regarding the nature of the information which will be stated orally or otherwise made available to potential subjects prior to their volunteering.

N/A