Integrative Health Systems, LLC

"One Cell One Light"©

To: [redacted]

H. Staninger © Feb. 7, 2010
Phone: [redacted]
Physician: R[redacted] B. M[redacted], MD

TOXICOGENOMIC DYSFUNCTIONAL ANALYSIS™ REPORT

Introduction:

The information contained in this report is for research purposes into the field of Toxicogenomics, which is the merger of toxicology and genetics and the association in the initiation of toxicological disease mechanism from exposure to environmental stress factors, weather modification, Aerosol Geo-Engineering, Project Earth Scope, BioTherapy, Genetic Therapy, Viral Vectors, Plasmids, Zinc Fingers Protein, Phasoms and many other technologies that are associated with exposure to a non-common factor in within the human meilue.[1]

The use of the HLA genes as addressed in this report are being utilized for the expression of a "mutation" in the gene loci, which is associated with the above types of exposures. These specific set of genes are used for organ typing, stem cell research, function and regulation of Cullin-Ring Ligase, SRF Proteins (actin regulation of calcium & hydrogen regulation of zinc fingers), and PLos Neglected Transfer Diseases[2] for virus amplifications of the following potential factors:

- Vitamin D receptors (VDR)
- Fcy receptor II (Fcy R II)
- Interleukin-4 (IL-4)
- Interleukin-1 receptor antagonist (IL-1RA)
- Mannos-binding lecitin (MBL)

HLA Class I & II gene molecules on infected cells with specific PLos Neglected Transfer Diseases, such as Dengue virus like other members of the Flaviviridoe family or other viral factors; increases the expression of these genes. HLA – controlled immune response may be responsible for the *immunopathy* of DV infections. Specific codons are 9 (any variance), 45 (Lys:aaa HIV – through SMV), 63 (prion codon 129/myelin PO protein precursor), 66 (transmechanism Cow/human through Zinc Finger Protein 66), 67 (protease position – Cys 67 and/or Aspartain 67). Untangling the genetic effects of "Codon Mutations and Amino Acids" for 67 is through polio virus reducing risk of causing attenuation of viral virulence by specific infectivity.[3]

Current, biotechnology pharmaceutical applied research in gene therapy utilizes Zinc Finger Protein as the therapeutic relevance of switching endogenous gene expression "on and off" at command inner and outer protein membranes. (Example: Cell membrane utilization of copper (1 molecule) and zinc (2 molecules) for superoxide dismutase (SOD-1) and the increase of C-protein (inside cell membrane) and G-protein (outside cell membrane).[4]

Many new developments in the last decade have opened the door for molecular biology and bioengineering through the commercialization of biotechnology. Codon(s) is a term used in molecular biology that allows DNA molecules to be placed at specific locations within the molecule of the subject matter. The codon(s) is its location to add mutations, additional DNA/RNA or many other plasmids, bacteriophages, ortholytics, proteins, zinc fingers, ring finger protein and specific cloned DNA of other species relative DNA information on its amino acid or protein.

Codon positions are 9, 45, 63, 66, and 67. Codon 66 is for transmerkinase cow human through zinc finger protein at positions 66. This position is used for the following species: *R. norvegius* *M. musculas*, *H. sapiens*, and *X. laevis* cloned tissue of adrenal glands, mixed tissue, ovary, heart and embryonic tissue. TRA-1 for the *Caenorhobditis elagones* and Drosophila ADR-1 and Sp1.[5]

Nuclear pore anchor, the Arabidoposis homolog of Tpr/M1p1/M1p2/megator is involved in mRNA export and samo homeostasis and affects diverse aspects of plant development. In some case of bioengineering the Arabidoposis homolog is used as a binding agent between Codons and the Tran located oligionucleotides, plasmid or other add ons.

The function and regulation of Cullin-Ring Ligases has been observed by Methae d. Petroski and Raymond J. Desholes work with the *Saceharomyces ceravisiae* and *C. elagones* through limb repatterning and the use of p53 – cytoplasmic anchor protein (PARC). Cullin is a family of proteins that are characterized by the presence of a distinct globule c-terminal clones. Through Cullin-Ring ligases basal and zinc involving metallothiosein resistance to cadmium via SOD-1's reaction with zinc fingers, zinc clusters and zinc twists in the DNA molecule. Zinc finger protein is currently being used in personalized HIV therapy DNA emerase to the CCR5 gene to cut a particle through smart dust applications in nanomedicine at the University of Pennsylvania and through Biomet microfixation as developed by the Stryker Corp for the Novartis Vaccination development in the Emeryville, CA facilities. The application of this type of technology reacts with C-protein (protein on the inside of the cell membrane) and can cause Fanconic anemia group C, while zinc finger via SOD-1 can cause a lack of trytophane 32 that potentates the toxicity of copper.[6]

Codons in rare cases, certain specific proteins may use alternative initiation (start) codes not normally used by that species (9) in certain proteins or non-standard amino acids like ethanol based amino acids.

Genetic Codes for nucleopolyhedrovirus UAG=Amber; UGA=Opal (umber); and UAa=ochre.

Hydropathy- grouping of codons are done by amino acids, residual moles volume and hydropathy.

The amine acryl-tRNA synthetases codons are leucine (UUA, CUU, CUC, CUA, CUG) and serine (UCA, UCG, UCC, UCU, AGU, and AGC). The release factors for codes on mRNA are initiation factors for prokaryotic, archeol (engineered) and eukaryotic cell structures.

Resent studies on the human gene HLA-B plays an important role in the response to HIV-1 infection, and different variants are strongly associated with the rate of AIDS progresson. It is therefore likely that different HLA-B alleles impose selection pressure on HIV-1, while HLA-B gene frequencies in the population are likely to be influenced by HIV (Nature, vol. 432, p. 769). This would be a symbiogenesis action of a virus.

RESULTS:

The following results are for Mr. K[redacted] A. P[redacted] HLA Class I A, B, C DNA and Class II DR, DQ DNA test results. Copies of the results that were furnished through Quest Diagnostics, Inc. are attached. The tests were performed using PCR and hybridization with sequence specific oligonucleotide probes (SSO).

HLA-A* 01 **HLA-A** are a group of human leukocyte antigens (HLA) that are encoded by the HLA-A locus on human chromosome 6p. The HLA genes constitute the major histocompatibility gene complex (MHC) of humans. HLA-A is a component of certain MHC class I cell surface receptors microforms that resides on the surface of all nucleated cells and platelets. The receptor is a heterodimer, and is composed of a heavy, α chain, and smaller beta (β) chain. The alpha chain is encoded by a variant HLA-A gene and the beta chain (β_2-microglobulin) is composed by the invariant Beta-2 microglobulin gene. **A (-) indicates homozyosity or unidentified allele.**

MHC Class I molecules are part of a process that presents polypeptides from host of foreign derivation to the immune system. Normally if a peptide of foreign, pathogenic, source is detected it alerts the immune system that the cell may be infected with a virus, and thus target the cell for destruction.

For humans, as in most mammalian populations, MHC Class I molecules are extremely variable in their primary structure, and HLA-A is ranked among the genes in humans with the fastest evolving coding sequence. After typing millions of individuals, hundreds of variant alleles and isoforms have been identified. This level of variation on MHC Class I is the primary cause of transplant rejection, as random transplantation between donor and host is unlikely to result in a matching of HLA-A, B or C antigens.

The HLA-A gene is part of the Human MHC complex on chromosome 6. The region is at the telomeric end of the HLA complex between the HLA-G and HLA-E genes. HLA-A gene encodes the larger, α-chain, constituent of HLA-A. Variation of HLA-A α-chain in certain ways is key to HLA function. This variation promotes diversity of class I recognition in the individual and also promotes genetic diversity in the population. **This diversity allows more types of foreign, virus or cancer; antigens to be 'presented' on the cell surface, but also allow a subset of the population to survive if a new virus spreads rapidly through the population.**

P. falciparum sporogaeti is linked to HLA A* 01 in Europe. A1 more common advent in people of Northwest Europe. Haploty is A1-Cw7-B8 (its serotype) specific for Irish

people from Ireland. Ancestral gene (A1 B8-DR17 (3) (DQ 2.5) East Africa. These individuals are prown to coeliace disease risks. A*0101 prown to type 1 diabetes. Class II diabetes linked to DR 3. B58 antineutrophil aystopassive antibodies (ANCA).

Chemici D.E. states that A25 is a split antigen of the A10 broad antigens of Western Europe in 95 % of the population and A10 in 2 % of the population. A2501 is high in Saudi Arabians.

HLA-A* 02 (A2) is associated with spontaneous abortions (females) and a higher viral load in HIV individuals with a Cw* 16 gene mutation.

HLA-A* 26 (2A6) is a human leukocyte antigen serotype within HLA-A serotype group. The serotype is determined by the antibody recognition of α^{26} subset of HLA-A α-chains. For A26, the alpha "A" chain are encoded by the HLA-A*26 allele group and the β-chain are encoded by B2M locus.[11] This group currently is dominated by A*2601. A26 and A*26 are almost synonymous in meaning. A26 is a split antigen of the broad antigen serotype A10. A26 is a sister serotype of A25, A34, A43, and A66.

A26 is more common in West Pacific Rim (Taiwan to Hokkaido). It is found in the transmembrane receptor/ligand.

HLA-B* 51 (B51) is associated with Behçet's disease, in endemic (versus epidemic) mucocutaneous lymph node syndrome, susceptibility to the virus that causes German measles infection.

Adamanitiades-Behçet's disease (ABD) is an inflammation of the wall of blood vessels that can involve the eyes, skin, and the rest of the body. Several alleles of B51 (B*5101, B*5108, B*5105, and B*5104) are found in disease, and linkage to markers, D6S285, in the HLA locus was strong (P>0.005). Homozygotes of B51 showed considerably high risk for disease indicating a possible gene-dose effect. B51 is capable of distinguishing several varieties of disease. HLA-B51 is found more frequently in disease that has an eye involvement. However it is less common in some regions when there is increased neurological involvement. The MICA*009 allele has been found to also associated with ABD when B51 is also present, IL-8 and other cytokines may also be involved. Sister chromatid exchange has also been observed more frequently in B51 (+) ABD.

However, B51 tends not to be found in ABD when a certain SUMO4 gene variant is involved and symptoms appear to be milder when HLA-B27 is present.

HLA-B* 55 (B55) is an HLA-B serotype. B55 is a split antigen from the B22 broad antigen, sister serotypes are B54 and B56. The serotype identifies the more common HLA-B*55 gene products. HLA-B* 59 is a hybrid of HLA-B* 55 and 51.

Mr. P[redacted] has both HLA-B* 51 and 55 but not 59.

HLA-CW*01 (C1) is associated with multinodular goiters. If Cw 16 B were present chronic lymphocytic leukemia. Cw 01 through HLA – E is restricted recognition of cytomeglio virus on CD 8+ cytolytic T lymphocytes.

HLA-CW*negative- is an HLA-C allele-group. The serotype identifies the more common HLA-Cw*negative gene products to natural killer cells with different amino acid sequences.

HLA-DRB1*03 (DR17) - is a HLA-DR serotype from Western Ireland, N. Spain and Sardinia. It is non-chronic sarcoidosis (arthritis of spine), infantile spasms/epilepsy. Rabius vaccine induced autoencephalomyelitis and cardiovascular hypertrophy in subjects with arterial hypertension. People with DR 17 show a tendency toward benzylpenicilloyl/allergies.

DRB1-0301 diabetes mellitus type 1, myositis, early onset Graves disease, type 1 auto immune hepatitis, inflammatory inclusion body myositis. In autoimmune hepatitis, DR B1-0301 correlates with more severe and difficult to treat disease.

HLA-DR17 is genetically linked to DR 52 and HLA-DQ2 serotypes. These serotypes are the result of gene products from the HLA-DRB3* and HLA DQA* 0501 and HLA DQB1* 0201 alleles. DRB!*0301 is frequently within the "Super –B8" or ancestral HLA haplotype A-0101: CW*0701:B*0801:DRB1*0301 DQA1*0501:DQB1*0201. This haplotype is known as "Super B8"; European ancestral haplotype or AH8.1

HLA-DRB1* 04 (DR4) - is a HLA-DR serotype effects extra articular rheumatoid arthritis hydralagine enilired female systemic lupus erythematosus, digestation's, phemphigus folliacusis, obstructive hypertrophic cardiomyopathy IgA nephropathy and shared syndromes systemic sclerosis/rheumatoid arthritis. Risk associated with increased risk for alopecia areata. DR 53 further suggest that the HLA system is one of the components of genetic susceptibility to leukemia but mainly in childhood and in boys only.

HLA DRB 5 (DR 51) is a HLA-DR serotype that recognizes the antigens encoded by the minor DR locus HLA-DRB5 and having distinct evolution having diverged from DRB1 approximately 4 million years ago.

DRB5 locus is only apparent in a small subset of DR haplotypes, and most individuals lack DRB***7)*** DRB3, DRB4, and DRB5 are minor DR beta encoding loci, they have been recognized as - is an HLA-DQ serotype that recognizes the common HLA DRB1*0301 and the less common HLA DRB1*0304 gene products. DQ7 is a form of 'split antigen' of the broad antigen group DQ3 which also contains DQ8 and DQ9.

DQ7 is linked by haplotype to a number of DQA1 (DQ alpha chain) genes, producing in cis-haplotype form, a large number of DQ αβ isoforms. These DQ alpha chains are also known to form transhaplotype isomers with other HLA-DQ.

DQ7 is linked to the following alpha chains genes (DQA1*). The serotyping efficiency of DQ7 toward DQB1*0301 is reasonably good, but still results in some false negatives, for *0304 the typing efficiency is poor and cross-reaction with DQ8 is relatively high. DQB1 03 shows increased mucosa lamerji and dry radiation and are at greatest risk for severe complications of coeliac disease, refractory disease, enteritis associated T-cell lymphoma (EATL)

Mr. K[redacted] P[redacted] did have a 17 amino acids lacking from his biological monitoring tests out of 22 possible amino acids. His Lyme KD - protein band was reactive for

P-41. The KD - protein band Lyme disease test in his other Advanced Biological Monitoring tests as not positive for any other protein band.

It is important to point out the protein gene band of P-39 gene deletion; which has been used in making a live *Brucella abortus* vaccine strain for cattle and sheep. B-39 protein is one of the major components of allergen manufactured by Rhone-Merieux. Lyon, France (brucellergene). A brucellergene fraction containing the P-39 induced a positive delayed hypersensitivity (DTH) reaction in infected guinea pigs and stimulated the production of IFN-gamma by blood cells of infected cattle in the research conducted by Anne Tibor, et. al. in 1998.[6] Due to the presence of other genes that are related to some form of viral protein envelop designed technology, if anyone has a reactive protein positive, then they may have a positive P-39 protein due to the technology utilizing the brucellergene in its nanotechnological architecture.

Mr. P[redacted] was reactive for KD-41, which is specific for exposure to Bubonic plague, thus individuals whose ancestors lived through the Middle Ages and exposure to Bubonic plague.

Conclusion:

The HLA Class I and II genes have shown specific mutations that may induce the specific diseases identified, if proper nutrients and other factors are not utilized to protect the body from expressing the codes of these mutations. It is very important to note that many of the HLA mutations identified for this research purpose illustrate possible exposure to specific gene vectors that are associated with expressing specific diseases through diagnostics tools and/or therapies. This is better addressed through specific codons utilized in the MIF Bioinformatics Databases EPIMHC HLA Data Base EpiTox Date PLoS Neglected Tropical Disease base for protecting and enhancing HLA alleles, University of California, Davis, CA.

The more mutations the greater the risk of expressing that genes mutated factor, but one must remember that through mutation a mightier hand than man's may be involved in taking the mutation and allowing it to hybridize and then "adapt" into beyond survival mode for the posterity of that individual, especially if they have high proteinoid expression for telomerase. In simpler terms, Mr. P[redacted] had specific HLA gene mutations that are related to Ireland's ancestral gene bloodlines.

If you should have any additional questions or concerns, please contact Integrative Health Systems, Dr. Hildegarde Staninger, RIET-1 for additional information.

I certify this report to be true and accurate to the information contained within and to the attached documents from Quest Diagnostics, Inc.

Signature: _____ Date: 2/7/2010

Dr. Hildegarde Staninger, RIET-1
Industrial Toxicologist//IH & Doctor of Integrative Medicine

REFERENCES

1. www.biotechnology.com

2. Lan, Nguyes Thi Phuony, et. al. Protective and Enhancing HLA Alleles, HLA-DRB1*0901, & HLA A*24 for Severe Forms of Dengue Virus Infection, Dengue Hemorrhagic Fever, and Dengue Shock Syndrome. PLOS Negl Trop Diseases. 2008 Oct: 2(10) e304

3. www.HLAClassI&IIDATABank.com and www.wikipedia.com HLA genes

4. Staninger, Hildegarde. Superoxide Dismutates. KACT – Korean Medical Association. Seoul, Korea © March 27, 2005

5. www.wikipedia.com Stryker Corp, Novartis Vaccine, Zinc Fingers, Cullin-Ring. © 4/19/2009

6. IBID ww.wikipedia.com BioMet, Inc., Jacksonville, Fl © 4/19/2009

7. Tibor, Anne, Jacques, Isabelle, Cuilloteau, Laurence, Verger, Jean-Michel, Grayon, Maggy, Wansard, Valerie, and Jean-Jacques Letesson. *"Effect of P-39 Gene Deletion in Live Brucella Vaccine Strains on Residual Virulence and Protective Activity in Mice."* Infection and Immunity. Nov. 1998, p. 5561-5564. © 1998 American Society of Microbiology.

8. Ryan, Frank. "I, Virus: Why you're only half human. New Scientist.com 29 January 2010 Magazine issue 2745.

Quest Diagnostics

QUEST DIAGNOSTICS INCORPORATED
CLIENT SERVICE 800.631.1390

PATIENT INFORMATION
P[redacted],K[redacted] A

DOB: [redacted] AGE: [redacted]
GENDER: M

ID: [redacted]
PHONE: [redacted]

REPORT STATUS FINAL

ORDERING PHYSICIAN
[redacted], D.O.

CLIENT INFORMATION
[redacted]

SPECIMEN INFORMATION
SPECIMEN: K[redacted]
REQUISITION: T[redacted]

COLLECTED: 01/12/2010 11:45
RECEIVED: 01/12/2010 22:42
REPORTED: 01/20/2010 10:46

Test Name	In Range	Out of Range	Reference Range	Lab
LYMPHOCYTE SUBSET PANEL 1				TBR
CD3%		56 L	57-85 Percent	
CD3 ABS		728 L	840-3060 Cells/mcL	
CD3+/CD4+ (HELPER)	44		30-61 Percent	
CD3+/CD4+ (HELPER) ABS	551		490-1740 Cells/mcL	
CD3+/CD8+ (SUPPRES)	12		12-42 Percent	
CD3+/CD8+ (SUPPRES) ABS		146 L	180-1170 Cells/mcL	
CD4/CD8 RATIO	3.77		0.86-5.00 Ratio	
CD19%	23		6-29 Percent	
CD19 ABS	314		110-660 Cells/mcL	
CD3-/CD16+CD56+ NK	18		4-25 Percent	
CD3-/CD16+CD56+ NK ABS	247		70-760 Cells/mcL	
ABSOLUTE LYMPHOCYTE CT	1352		850-3900 Cells/mcL	
HLA A,B,C CLASS I DNA TYP				AMD
HLA-A*	02(A2)			
HLA-A*	26(A26)			
HLA-B*	51(B51)			
HLA-B*	55(B55)			
HLA-Cw*	01(Cw1)			
HLA-Cw*	-(Cw-)			
RESULTS REVIEWED BY	see note			

William W. Ward, Ph.D., D(ABHI)

Serologic equivalent is given between parentheses.

The (-) indicates homozygosity or unidentified allele.

Typing performed by PCR and hybridization with sequence specific oligonucleotide probes (SSO).

Toxicogenomic

PERFORMING LABORATORY INFORMATION:

AMD Quest Diagnostics Nichols Chantilly 14225 Newbrook Drive Chantilly VA 20151
Laboratory Director: Kenneth Sisco, MD,PhD CLIA No: 49D0221801

TBR Quest Diagnostics One Malcolm Avenue Teterboro NJ 07608 Laboratory Director: William E. Tarr, M.D.
CLIA No: 31D0696246

Quest Diagnostics®

QUEST DIAGNOSTICS INCORPORATED

PATIENT INFORMATION
P[redacted],K[redacted] A

REPORT STATUS **FINAL**

DOB: [redacted] AGE: [redacted]
GENDER: M

ORDERING PHYSICIAN
[redacted], D.O.

SPECIMEN INFORMATION
SPECIMEN: K[redacted]
COLLECTED: 01/04/2010 12:06
REPORTED: 01/13/2010 10:45

CLIENT INFORMATION
[redacted]

Test Name	In Range	Out of Range	Reference Range	Lab
C5,SERUM	17.6		6.0-20.0 mg/dL	AMD
Low levels of C5 indicate either increased catabolism or decreased synthesis.				
CHOLINESTERASE,RBC/PLASMA	Test Not Performed			TBR
No acceptable specimen was received				
HLA-DRB 3,4,5 DNA TYPING				AMD
HLA-DRB	4(DR53)			
HLA-DRB	5(DR51)			
RESULTS REVIEWED BY	see note			
William W. Ward, Ph.D., D(ABHI)				

Toxicogenomics

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FOOTNOTE(S):

1 This test was performed using the Siemens (Bayer) chemiluminescent method. Values obtained from different assay methods cannot be used interchangeably. CEA levels, regardless of value, should not be interpreted as absolute evidence of the presence or absence of disease.

2

This test was performed at:
Focus Diagnostics, Inc.
5785 Corporate Avenue
Cypress, CA 90630-4750

PERFORMING LABORATORY INFORMATION:

AMD Quest Diagnostics Nichols Chantilly 14225 Newbrook Drive Chantilly VA 20151
Laboratory Director: Kenneth Sisco, MD,PhD CLIA No: 49D0221801

ARL Focus Diagnostics, Inc. 5785 Corporate Avenue Cypress CA 90630 Laboratory Director: Alfred Lui, MD
CLIA No: 05D0644251

KD PROTEIN BAND MARKERS MEANINGS

KD Band No.	Marker Meanings
18	Translocater protein target for Anxiotytic (drugs) Binds to plant chlorophyll.
23	Calcium oxalate (21 to 25 KD).
23 & 41	Late Chromium migraines & rash as found in Lyme - No IgG.
41	Bubonic plague
21 to 25	Calcium oxalate monohydrate binding. 21 – tyrosine-phosophorylated
24	Tyrosinogen "parasitism"- specific hemolymph protein (sugars). Concanavalin A (CON A)
28	Cochlear protein – Menieres disease
30	Cornea protein
39	Plasma membrane protein & anchor for glycopphorin "oxtyl glycoside"
41	Bubonic plague "Yersinia pestis" 15 – same
45	Simian Monkey Virus (cancer initiator)
47	Canine herpes virus
58	Monocytic membrane protein (+ 78 KD) – Floral organs 91, 87, 58, 53, 37, 30 -26
66	Heat Shock Proteins
93	Membrane vesicle on surface of B. Burdorferia (crystal) Increases CD 43 that regulates tyrosine phosphoralate
80	Glycoproteins
31	Lack of human cumulus – cornea radiation
28 & 30	Cochlear Protein Antibody
70	Heat Shock Protein
50	Enhanced glycosylation
27, 34, & 41	Plant subunit vaccine – pneumonia & Bubonic plague

	Bubonic pneumonia virulence Caf1 gene Anti Yad C antibody YadBC F1 Protein Capsule. Bubonic Plague antibody pH6 with KD 30, 43, 60 & 75.
26	UV induced RNA protein crosslinks in snRNPs (covalent bond crosslinks)
58	Calmodulin – binding glutamate decarboxylase organ specific expression of thyroid hormone receptor mRNA TRbeta1 (+55 & 52 KD) CiNii
	-inner ear disease in Guinea pig. 58 Kd CE-Binding protein with 30.
	- Rat pancreas Golgi subfraction pancreas B2 proteins with anti B 2
170, 152, 139, 120 88, 65.5, 58 & 45	White Spot Syndrome Virus

Additional Special Notes

KD	**Meaning**
71 – 61	**Spirochete Bacteria** Leptospirosis – Weil's Fever Rat Bite Fever – Sodoku (Japan) *Streptobacillosis moniliformis* Haver Hill Fever Relapsing Fever (includes relapsing fever of Lyme disease) – Typhinia – *Borrelia recurrentistidi* *Borrelia hermsii* *Borrelia parkeri* *Ttepponematoses* – Gonorrhoea (veneral disease/Syphilis)
	Syphilis – can compromise the following organ systems Syphilis Mexican Bladder, Cardiovascular, Congenital, Cryptogenic, Digestive, Ear, Eye, General, Joints, Lymphatic, Nerve, & Teeth

Quest Diagnostics

QUEST DIAGNOSTICS INCORPORATED
CLIENT SERVICE 800.631.1390

PATIENT INFORMATION
P[redacted], K[redacted] A
DOB: [redacted] AGE: [redacted]
GENDER: M

REPORT STATUS FINAL

ORDERING PHYSICIAN
[redacted], D.O.

SPECIMEN INFORMATION
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REQUISITION: T[redacted]

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[redacted]				
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HLA A,B,C CLASS I DNA TYP				AMD
HLA-A*	02(A2)			
HLA-A*	26(A26)			
HLA-B*	51(B51)			
HLA-B*	55(B55)			
HLA-Cw*	01(Cw1)			
HLA-Cw*	-(Cw-)			
RESULTS REVIEWED BY	see note			

William W. Ward, Ph.D., D(ABHI)

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