THE UNIVERSITY FOR DEVELOPMENT STUDIES

STATISTICAL ASSESSMENT OF FADING FINGERPRINT

BY
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DEDICATION

To my wife Winifred Boahen and children Emmanuel, Bright, Christy and Rhoda.

DECLARATION

I hereby declare that this thesis is the result of my own original work and that no part of it has been presented for another degree in this University or elsewhere.

ERIC BOAHEN 26-06-11

Signature Date

Supervisor's Declaration

I hereby declare that the preparation and presentation of the thesis were supervised in accordance with the guidance on the supervision of thesis laid down by the University for Development Studies.

Principal Supervisor
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Signature Date

Co - Supervisor ....

Signature Date

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Abstract

Fingerprint emerged as an important system within the security agencies, government offices and court of law in the late 19th century, when it replaced anthropometric measurements as a more reliable method for identifying persons. No two fingerprints have ever been found identical in billions of human and automated computer comparisons. Fingerprint is thus claimed to outperform DNA and all other human identification systems. However, genetic mutation and effect of certain drugs have been found to influence changes in some of the fingerprint features but the extent has not been modeled adequately. This study is aimed at studying these potential differences from the effects of drugs. In the study, sample data in the form of patient's fingerprints are transformed to quantitative data for statistical analysis. Two statistical approaches Gen Stats analysis and stochastic) are used. For the stochastic approach, we describe absolute changes in fingerprints as function of selected drugs and covariates patients' age and duration of drug use. Fading fingerprint models for cancer chemotherapy are described as optimal control problems and the maximum level of toxicity store in the normal cells is represented by $P_I(\Delta x) = 1 - [1+ e^{\beta 0}][1 + e^{\beta 0 - \beta \Delta x}]^{-1}$ and this measures the swelling and expansion of the palm and consequently the peeling of ridges. We also discuss optimal therapies when the controls represent the effectiveness of chemotherapeutic agents, or, equivalently, when the simplifying assumption is that drugs act instantaneously. In addition to this, we describe the intensity of cancer with $w_I(t) = 2r_1r_2Noq^2$,the level of damage done to DNA and PCR with $\int_0^t w_1(u)du = 2r_1r_2q^2fN(u)udu$ where drug usage is zero using stochastic models, based on biological processes predicting future results in fading fingerprint. We further established that the growth of cancer may be represented by $x = (1-q)\beta s-(1-r)\beta r+\sqrt{((1-q)\beta s-(1-r)\beta r)^2+4rq\beta s\beta r} > 0$, where x is the ratio of sensitive killed-cells (S) to the resistant developed cells, R *(that is,* $x = s/R$). Thus, left alone, cancer cells grow exponentially reaching the relative proportions $S = \bar{\bar{x}}R$. This study has raised important medical issue of drug resistance and the maximum level of penalty in drug usage beyond the resistant stage. The effect of cancer drug model discussed here predicted the clinically established dandelion phenomenon and suggested depleting ridges by cancer drugs. The implication arising from the study suggests the need to avoid absolute reliance on fingerprint for identification and financial transactions. Consequently, it is recommended that

a policy be put in place to monitor and review fingerprint features of cancer patients, and to incorporate other biometric characteristics (e.g. eye, gait) for purposes of identification.

List of tables

List of figures

List of symbols page first used

Symbol	Explanation	Page first used
	Less than	14
<	Less than or greater than	14
=	equal to	14
$\sum_{i=1}^{m} u_i B_i$	sum of uB from i=1 to m	15
∪	Union of	15
α	alpha	15
	Beta	15
∞	Infinity	15
≥	greater than or equal to	15
>	greater than	15
∫	integral	15
Δ	change	21
ε	error term	22

List of abbreviations

Abbreviation	**meaning**	**page used**
AK	**Alkylating**	**22**

List of abbreviations

Abbreviation	meaning	page used
AK	Alkylating	22
ATM	Antimetabolic	22
OT	Others	22
NP	Natural product	22

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1.0 INTRODUCTION

1. 1 BACKGROUND OF THE STUDY

It has been claimed that the ancient scientist were aware of finger print and used a kind of fingerprint system thousand years ago. Before that, other anthropometric systems of identification such as hair color or style, weight or eye color were used, these may change in time. In earlier civilization, branding, tattooing or even maiming was used to mark and identify criminals. Although man had been aware of the fact that each individual possessed a unique set of ridges on the hands, the use of these prints for criminals identification was not accepted until 1900's. It was an English assistant commissioner of the Metropolitan police force, Sir, Edward Henry who invented a standardized means of classifying human fingerprints which has been used throughout most of the world. The Ghana Police Service (Criminal Investigation Department), Bureau of National Investigation (BNI), Immigration Service and many others use fingerprint mostly in identifying people. This enormous collection is composed of both criminals and civil prints .The civil files include the prints of both government employees and applicant for national jobs.

The validity of forensic fingerprint evidence has been challenged recently by academics, judges and the media. While fingerprint identification was an improvement over the years, the subjective nature of matching, despite a very low error rate has made forensic practice controversial. Certain specific criticisms are now being leveled against by some leaders of the forensic community providing an incentive to improve training and procedures. Glenn Langenburg, who is a Forensic Scientist, Latent Print Examiner for the Minnesota Bureau of Criminal Apprehension, is such an individual, having written an article that responds to the most academic critics (www.en.wikipedia.org/wikilFingerprint). The word reliability and validity have scientific meanings to the scientific community. Reliability means successive tests bring the same result of an individual fingerprint with time. Validity means that the results accurately reflect the external criteria (minutiae) being measured. Althoūgh experts are often more comfortable relying on their instincts, this reliance does not always translate into superior predictive ability due to the effect of cancer drugs. Despite the absence of objective standards, scientific validation, and adequate statistical studies, a natural question to ask is how well

fingerprint examiners actually perform to substantiate consistency of the same individual fingerprint over time. (www.en.wikipedia.org/wikilFingerprint, 2008).

Experts' testimony based on fingerprint evidence is delivered in a courtroom by comparing salient features of a latent print lifted from a crime scene with those taken from the defendant. A reasonably high degree of match between features is expected to lead the experts to testify irrefutably that the source of the latent print and the defendant are one and the same person. For decades, the testimony of forensic fingerprint experts was almost never excluded from these cases, and on cross examination the foundations and the basis of this testimony were rarely questioned. Central to establishing an identity based on fingerprint evidence is the assumption of discernible uniqueness. Salient features of successive prints of the same individual are observably the same. The assumption of discernible uniqueness, although lacking sound theoretical and empirical foundations, allows forensic experts to offer an unquestionable proof towards the defendants' guilt (Yongfang Zhu, Sarat, C. Dass, and Anil, K. Jain, 2008). To make matter worse, forensic experts are usually not questioned on the uncertainty and natural variations associated with their testimonials.

The fact that sequence of nucleotides in our DNA is as individual as our fingerprint, the basis of a new method of identification depends on natural conditions. The effect of DNA damage on replication and how frequently would an observable match between a pair of prints lead to errors in the identification of individual with time has been overlooked by experts. Genetic profiling was first used in evidence in the late 1980's to establish the guilt of a murderer. Since then the technology has been the subject of intense scrutiny in courts of law and in the media, and the term genetic fingerprint was soon coined, but patterns on the skin or physical mapping of DNA has nothing to do with it at all since fingerprint (though unique) is subject to fading due to possible effect of certain drugs. The first case using genetic fingerprint proved a suspect who had confessed to a rape and murder was declared innocent based on forensic interpretation. Two girls had been raped and murdered in the same part of the country, but the crime had been committed three years apart. Investigators suspected a connection between the two and soon a suspect was cross examined. The suspect admitted to one of the crimes but denied being

involved with the other charge. Forensic scientists were called to analyze the latent fingerprint lifted at the crime site and the fingerprint of the suspect was equally taken and both analyzed and compared. Interestingly, the result exonerated the suspect who had initially admitted to one of these charges. It would soon be revealed that the suspect was a cancer patient and cancer drug had affected minutiae pattern analysis in his fingerprint. Blood sample at the scene of the crime were picked and DNA profiling was later resorted to which showed clearly that both crimes had indeed been committed by the same suspect (Jenkins et al., 2003). The analogy is the uniqueness of both set of fingerprint and a DNA profile. The technology of using biometric fingerprint is particularly good for identifying culprits of violent crimes without natural variations.

Several works had already been done on fingerprint, among these; some express the existence of genetic fingerprint, individuality of fingerprint, analysis and comparing fingerprint with verifying probabilities. None of these studies was able to gear towards the changing trend in genetic mutation and environmental degradation that influence genetic physical mapping of the DNA. Yet on consensus tool such fading fingerprint model has emerged. One of the reasons behind the lack of consensus is that there has been no systematic, large-scale open on genetic mutation and their statistical distributions on fingerprint. It is in the light of this problem, that we have taken the pains to investigate to prove the fading genetic fingerprint.

1.2 STATEMENT OF THE PROBLEM

Cancer drug erases and fades fingerprints, inflammation and blistering removes fingerprint. Commonly used cancer drug makes patients fingerprint for identification erases (http:/blogs.discovermagazine.com/discoblog/2009/05/27/cancer). A patient was recently held by United State of America immigration officials for more than four hours before he was allowed to enter the country upon investigating to find out that he was not a treat to security. His doctor, Eng-Huat Tan from Singapore explained that his client, 62-year old man had head and neck cancer but had responded well to capecitabine to help prevent the cancer coming back. (http://www.smh.com.au/worldldrug-erase, (2008/04/16)

Although the drug is commonly used to treat a range of cancers, it however cause chronic inflammation of the palm over time and this leads to loss of fingerprint.

(http://www.dailytelegrph.com.aullifestyllnews/cancer drug). Although, the effect of certain drugs has been found to influence changes in some of the fingerprint features, the extent has not been modeled adequately. This study is however, aimed at studying these potential differences from the effect of cancer drugs.

The e-zwich operates solely on fingerprint, and it is sensitive to any variation in the already stored information on fingers. In the process customers are identified with their fingerprints already stored so as to constantly carry out transaction. It is against the background of future variation and fading fingerprint that the system is adopted to store information on all the ten fingers even though, genetically, they contain the some information. Eventually, the system anticipated variation and fading in fingerprint as a result of external factors such as dirt and DNA damage, fingers are cleaned before prints are taken but variation and fading emanating from DNA damage due to effect of cancer drugs still remain unaddressed. I personally had an interview with a team of e-zwich officials who carried out registration exercise at the University for Development Studies-Navrongo campus, and it was confirmed that potential customers are sometimes denied of their own account.

1.3 SIGNIFICANCE OF THE STUDY

(1) The findings of the study are relevant to cancer patients who may have to use fingerprint in their day to day activities.

(2) The findings of the study are useful to institutions such as Bureau of National Investigation (BNI), Criminal Investigation Department (CID), Court of Law and other agencies to combat crime.

(3) This work is equally important to Biometric Passport Officials, National Identification Authority Officials, Immigration Officials and e-zwich agencies that operate fully on fingerprint as personal identification.

1.4 OBJECTIVE OF THE STUDY

An analysis of variation in fingerprint requires the development of appropriate statistical models on the space of fingerprint variation that are able to represent all aspect of genetic mutation. Statistical models of fingerprint are essential for a variety of task. For instance, fading in fingerprint can be viewed as a message produced by a stochastic source and understanding the statistical regularities of the source is essential for deriving the necessary algorithms that used short code words for the most frequent changes. In this work statistical models are essential in at least two main ways; (I) to model and approximate the distribution of variation scores. (II) to assess the significance against chance of fading where chance can be defined in several ways. The problem of establishing fading estimate based on fingerprint is in contrast to DNA typing error due to genetic mutation.

The act of acquiring fingerprint impressions as well as conditions of physical finger itself such as cuts, bruises and distortions as well as biological mutagenic and chemical physiology that permit fading ridges introduces several errors.

The study objectives may therefore be summarized as:

- To investigate the effect of cancer drugs on changes in patients finger print
- To find the probability of change in ridge pattern among cancer patients
- To estimate and compare the fading rate of major class of drugs used by cancer patients

2.0 LITERATURE REVIEW

2.1 Introduction

Some people regard fingerprint practice as a closed discipline. Existing works on fingerprint identification are centered on analysis and individuality.

Perhaps the most analysis method for assessing fingerprint is chromosome walking. In this case, a starter clone is used as a probe to isolate overlapping clones from human gene. Hybridizing clones are analyzed and those extending from the original clone are identified. A more recognized approach was developed by Birkenbihl and Vielmetter (1997). They hybridized

filters containing an array of E. coli clone using whole cosmid clones from the array as probes. The multiplex method used by Evans and Lewis is another adaptation of chromosome walking to an ordered array of clones. The principle is to hybridize probes derived from every clone with all others in the clone collection.

Instead of repeatedly rescreening a library, a number of alternative methods for overlap assembly have been devised. These allow large number of randomly clone picked to be analyzed and matched. A fingerprint for each clone is produced which can be stored in the database on computer. The software will then compare the fingerprint of a clone pair wise with all other clones in the database, looking for similarities. The method used by Olson et al.; (1997) for creating a *Saccharomyces cerevisiae* ordered library was to digest DNA from randomly picked clones with a combination of Eco RI and HindIII and separate the fragment by agarose gel electrophoresis. Fragments between 0.4 and 7.5kb in size were entered into the computer using a digitizer and the patterns generated by each clone compared pair wise with other clones. Coulson et al., (2001) followed a comparable approach in his work on *Caenorhabditis elegans*. However, the sensitivity of the procedure was improved by using the superior resolution of sequencing - style denaturing polyacrylamide gels and introduced greater automation for data input. He digested DNA from individual clones with HindlII and end-labeled with dATP using reverses transcriptase. After inactivating the enzyme, he performed a second digest with Sau3A, fractionated the DNA on a 4% denaturing polyacrylamide gel, dried and autoradiographed the gel. This produced a pattern of perhaps 20 bands on average, which was sized by comparison with marker track (DNA digested with Sau3A). The data on the autoradiograph were into the computer using a densitometric scanner. Each band was then accepted or rejected using interactive image analysis software in order to exclude artifactual band. As with Olson's method, each clones compared with each other clone and the probability of the match being due to chance was calculated. An automatic contig assembly program can be used where a threshold probability is given. However, it was recommended that fingerprints are also checked manually using the calculated probabilities as a guide.

Carrano et al., (1996) described a similar method, but they labeled the ends with fluorochrom thus allowing detection by an automated DNA sequencing apparatus, and hence facilitating direct entry of the data into a computer. Sample sequencing is a novel variation of Coulson's

work which has recently been suggested by Brenner and Livak, (2000). Both works by Carrano and Coulson are limited in that the only information recorded for each band is its size.

The maximum number of possible bits of information is therefore determined by the resolution of the gel. Although an automated sequencing machine was used, manual sequencing with radioactivity can also be used.

Instead of analyzing the patterns produced following restriction enzyme digestion of clones, Craig et al., (1998) fingerprinted clones by hybridizing short oligonucleotides to an array of cosmid DNA clone to a filter.

There have been few studies that addressed the problem of fingerprint individuality using statistical models on fingerprint features. All these studies utilized minutiae features in fingerprints (both location and direction) to assess individuality. However, the assumptions made in these studies did not satisfactorily represent the observed variation of the features. For example, it is known that fingerprint minutiae tend to form clusters but Pankanti et al., (2000) assumed a uniform distribution on minutiae location and direction which was then corrected to match empirical results from the database used in their studies. Another assumption made by Pankanti et al., (2002) is that minutiae location is distributed independently of the minutiae direction. However, minutiae in different regions of the fingerprint are observed to be associated with different region-specific minutiae direction. A minutia is the location of a ridge anomaly in a fingerprint.

Wu, Jin Chu and Michael (2006) worked on nonparametric statistical data analysis of fingerprint minutiae exchange with two finger fusion. The utility of this method was demonstrated through analyzing results from minutiae exchange with two finger fusion. They focused their analysis on high accuracy vendors and the two modes of matching standard fingerprint templates; native matching where the same vendor generates the templates and the matcher. They also introduced scenario 1 interoperability where vendor A's enrollment is matched to vendor B's matcher. The study demonstrated the utility of applying nonparametric inferential statistics to biometric test results. One main advantage of this approach was that, since there is no underlying distribution model for fingerprint data, the statistical analysis data could be modeled independently. This method is applicable on small sizes of samples and when the availability of samples is limited where the normal distribution cannot be applied. However,

the statistics invoked in their study were too inferential rather than descriptive. In this way, properties of the population were inferred from the sample which lack potentially descriptive insights with focus on individuals in the sample rather than inferred.

Several probabilistic models for developing drug effects on cells exist in the literature. For example, in one of the early classical work Codman and Goldie, (1983) the tumor size was analyzed as a stochastic process and the probability no resistance cell was maximized. More recently, a probabilistic model for the evolution of the drug sensitive cancer subpopulation from a single mutational cell was formulated and analyzed numerically Westman et al., (2002). Furthermore, a broad class of models which describe drug resistance not as a single mutation event, but as a branching process, has been developed ,for instance ,in Hamevo, Agur, Kimmel and Axelrod, (1991). Corresponding infinite-dimensional deterministic model have also been formulated and analyzed by Swierniak et al., (1995). However, due to the high dimensionality, these models often allow limited analysis. Serwa et al., (2010) worked on fingerprint of children under one year (Africa Centre Site). She demonstrated the feasibility of fingerprint -based individual identification for population based research in developing countries. According to her there is a record linkage between demographic surveillance population's database and healthcare facility data based on biometric identification. In her work, fingerprint of children under one year varied between 94.1% to 96.7% and by age 5, children fingerprint enrolment rates were comparable to those of the adults.

2.2 Cancer Drug and Effects

The objective of cancer drugs is to kill cancer cells with as little damage to normal cells. In malignant tumors, it is obvious to see many cancer cells being divided, so that many cancer drugs are designed to interfere with cell proliferation, often by blocking synthesis of DNA, RNA or protein. One major class of cancer drugs is that of the alkylating agent. This kind of drug binds to DNA and chemically modifies it, interfering with replication and transcription Tripathi, (2008). Chemically, methotrexate, a folic acid antagonist inhibits the enzyme dihydrofolate reductase and prevents transfer of methyl groups in biosynthetic reactions, including synthesis of deoxythymidine in DNA. The use of 5-Fluirouracil serves as an analogue of thymine and prevents DNA synthesis by inhibiting thymidylate synthetase. A limiting factor

in cancer drugs is its toxicity to normal tissues. If whole body could be exposed to 12,000 centi-Gray of ionizing radiation, every cancer cell would be killed Tripathi, (2008). Unfortunately such drugs would also kill the patient. Since the major constituent of living cells is water, exposure of them to ionizing radiation such as x-ray or v-rays will result in hydroxyl radical production Tripathi, (2008) .OH (hydroxyl) is responsible for a large part of the damage done to cellular DNA. Single and double strand breaks in DNA are considered to be very important damaging events. In this case, a predictive mathematical model for the effect of cancer drugs hypothesis is used to understand PCR responses to cancer drugs and judge the efficacy of cancer treatment. The importance of drug efficacies on different abnormal cell population is investigated through the kinetics associated with their effect to therapy, results and fading fingerprint. The model discussed here aims at predicting the clinically established dandelion phenomenon.

Recent models for cancer therapy are cell-specific and treat the cell cycle as an object of control. Each cell passes through a sequence of phases from cell birth to cell division. The starting point is the growth phase G_1 after which the cell enters a phase S (synthesis phase) where DNA synthesis occurs. Then a second growth phase G_2 takes place in which the cell prepares for mitosis or phase M. Each of these two daughter cells can either reenter phase G_1 or for some time may simply lie dormant in a separate phase G_0. These distinctions are important since most cancer drugs are active in a specific of the cell cycle. Drug treatment influences the cell cycle in many ways. The most fundamental aspect is cell-killing but also blocks other agents from playing reversal roles. Blocking agents slows down the transition of the cell through the cell cycle and thus impedes on the tumor's growth while recruitment agents make cancer cells leave the dormant stage G_0 where they are not susceptible to any drug.

3.0 METHODOLOGY

3.1 DATA

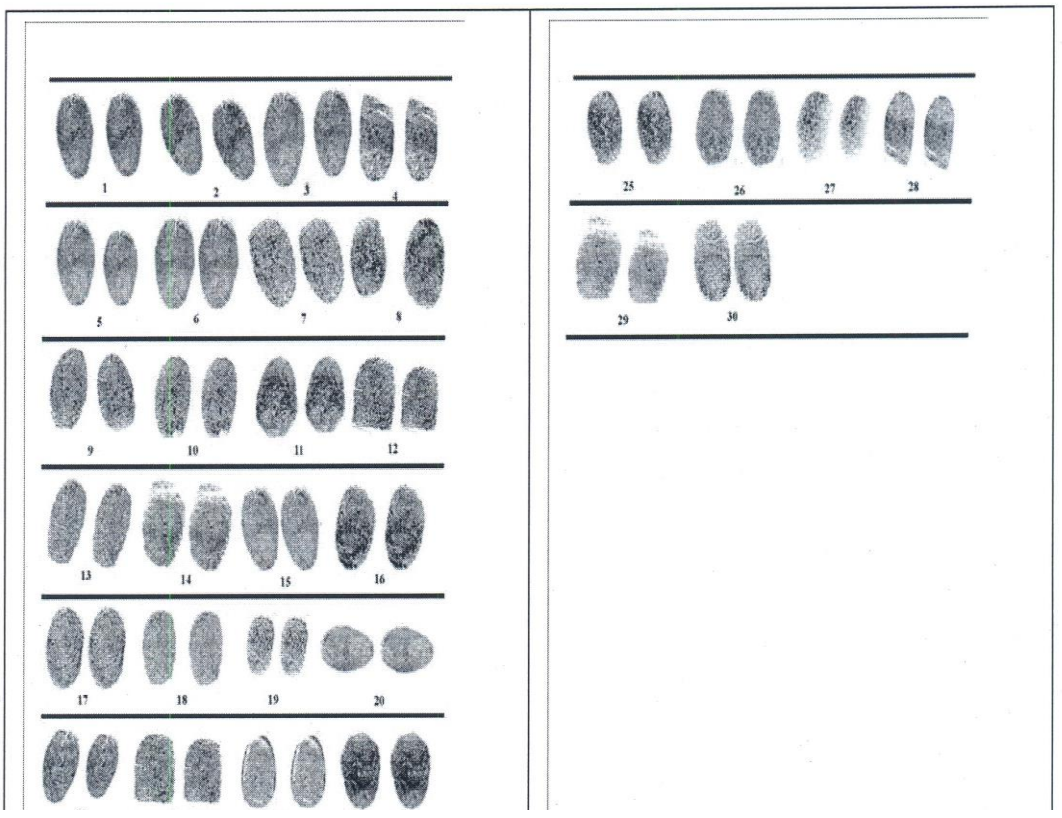
3.1.1 Patients selection and Fingerprint Data Collection

Fingerprint data for the study were obtained from cancer patients at Komfo Anokye Teaching Hospital, Kumasi Ghana. Participation in the test was voluntary. Patient's medical history such as the type of drugs used and method of medication were recorded. In addition to this, patient's

age, cancer duration, and drug use duration were recorded. Besides these variables, patient's fingerprints were taken and recorded by print A. The second fingerprints were taken after six months of the first fingerprint and denoted by B. The reason for taken the second fingerprint after six months interval was to see whether there would be change (fading) in patients fingerprint as they continue to use the drugs. This is resistance stage where sensitive cells are killed but resistant cells continue to exist and cancer treatment beyond this stage increases toxicity in the cells causing fading in ridges.

3.1.2 SAMPLE DATA

The sample data (Figure 1) collected (fingerprints) were scanned and enlarged so that spaces between ridges were visible. The resulted enlarged sample data was printed out for measurement. A pair of divider and ruler was used to measure distance between two successive ridges in the first fingerprint of individual (print A) and the results recorded. The process was repeated for the second fingerprint of all the patients (print B) to obtain quantitative data (Table 1).



Patients	Age t(yrs)	cancer(Agea)(yrs)	Intermittent(yrs)of drug used	Distance between ridges at first print (A)	Distant between ridges at second print(B)	Type of drug
1	52	5	4	0.096	0.140	melphalan
2	65	8	5	0.090	0.103	5-fluorouracil
3	42	4	3	0.082	0.135	Chlorambucil
4	40	7	4	0.096	0.116	Methotrexate
5	40	5	3	0.090	0.138	Busuphan
6	33	6	3	0.084	0.133	Cyclophosphamid
7	62	6	3	0.092	0.097	Cytosine arabinos
8	50	9	3	0.089	0.105	Cisplatin
9	49	10	4	0.094	0.119	Vinblastine
10	60	9	3	0.099	0.160	Bleomycin
11	57	7	4	0.101	0.133	Daunorubicin
12	45	5	3	0.097	0.141	Busulfan
13	67	9	6	0.089	0.099	6-mercaptopurine
14	75	8	4	0.095	0.104	Tamoxifen
15	43	5	2.7	0.099	0.142	Procarbazine
16	40	4	1	0.095	0.102	Vincristine
17	75	5	3	0.097	0.136	dicarbazine
18	56	9	7	0.089	0.127	6-thioguanine
19	55	7	2	0.097	0.132	Actinomycin D
20	34	6	3.6	0.104	0.165	Bleomycin
21	32	5	2	0.087	0.098	Mitomycins
22	54	4	3.2	0.097	0.109	Streptonigrin
23	52	7	3.1	0.130	0.174	Chlormbucil
24	77	8	4	0.098	0.130	Cisplastin
25	56	5	3.2	0.110	0.154	Tamoxifen
26	34	4	2.6	0.099	0.154	Adriamycin
27	63	4	3	0.099	0.164	Methotrexate
28	67	3	1.8	0.094	0.099	Dicarbazine
29	38	4	2	0.093	0.130	Melphalan
30	78	7	3.5	0.120	0.167	bleomycin

TABL 1: Quantitative data derived the fingerprints

The drugs indicated on table 1 are nested into four major classes of cancer drugs. This is necessary because most of the drugs have different trademark names but possess the same constituents which perform the same function. Drugs under each class perform similar function in treating cancer. These classes are Alkylating drugs which modify DNA, Antimetabolic drugs which interfere with DNA synthesis, Natural products which serve as antibiotic and Others

TABLE 2: Major class of cancer drugs used by patients in the research

KYLATING DRUGS	ANTIMETABOLICS	NATURAL PRODUCT(antibiotic)	OTHERS
Modify DNA)	(Interfering DNA synthesis)	doxorubicin(Adriamycin)	tamoxifen
hlorambucil	5-fluorouracil	daunorubicin	cisplatin
lelphalan	methotrexate	actinomycin D	dicarbazineprocarbaz
/clophosphamide	cytosine arabinoside	mitomycin C	procarbazine
usulfan	6-mercaptopurine	vinblastine	
osfamide	6-thioguanine	vincristine	
		bleomycin	

3.1.3 LIMITATIONS

To increase sample size was to increase precision and this involves cost. It was however not easy to get patients to participate in the test. Some had demanded payment for transportation, while the time frame or frequency for taking patients fingerprint was not adequate.

4.0 MODELING, APPLICATIONS AND RESULTS

Application and analysis of results

FIGURE 5:Ridge fading measured within six month interval

Modeling Change

. The empirical distribution is fitted in the regression model (equations 27, 28).

$$Y=\beta_0 + \beta_1 T + \beta_2 a + \beta_3 \Delta + \beta_4 V + \beta_5 d + \varepsilon \quad (1)$$

Where y= absolute change

T= patients age

a=cancer duration

Δ= change scores

V=class of drug

d=duration of drug use

4.1.4.1 Results - $Y=\beta_0 + \beta_1 T + \beta_2 a + \beta_3 \Delta + \beta_4 V + \beta_5 d + \varepsilon$

TABLE 3: Derived table indicating change scores and absolute change from table (1)

patient	AgeT	cancer_dur	drugUse_dur	change abs	Class of drug	Change scores
1	52	5	4	0.459	AK	
2	65	8	5	0.144	ATM	0
3	42	4	3	0.646	AK	1
4	40	7	4	0.217	ATM	1
5	40	5	3	0.533	AK	1
6	33	6	3	0.581	AK	1
7	62	6	3	0.054	ATM	0
8	50	9	3	0.179	OT	0

9	49	10	4	0.253	NP	1
10	60	9	3	0.516	NP	1
11	57	7	4	0.317	NP	1
12	45	5	3	0.453	AK	1
13	67	9	6	0.112	ATM	0
14	75	8	4	0.095	OT	0
15	43	5	2.7	0.434	OT	1
16	40	4	1	0.074	NP	0
17	75	5	3	0.402	OT	1
18	56	9	7	0.427	ATM	1
19	55	7	2	0.126	NP	0
20	34	6	3.6	0.587	NP	1
21	32	5	2	0.126	NP	0
						0
22	54	4	3.2	0.124	NP	
23	52	7	3.1	0.338	AK	1
24	77	8	4	0.327	OT	1
25	56	5	3.2	0.4	OT	1
26	34	4	2.6	0.556	NP	1
27	43	4	3	0.056	ATM	1
28	67	3	1.8	0.053	OT	0
29	38	4	2	0.398	AK	1
30	78	7	3.5	0.392	NP	1

GenStat output 1: The model considering all the variables (age, cancer duration, drug use duration, change scores, drug and change absolute)

```
***** Regression Analysis *****

 Response variate: change abs
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Fitted terms: Constant + AgeT + cancer_dur + change scores + drug + drugUse_dur

*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.	F pr.
Regression	7	0.7855	0.11221	10.73	<.001
Residual	22	0.2301	0.01046		
Total	29	1.0156	0.03502		

Percentage variance accounted for 70.1

Standard error of observations is estimated to be 0.102

* MESSAGE: The following units have large standardized residuals:

Unit	Response	Residual
9	0.253	-2.04
27	0.056	-2.21

*** Estimates of parameters ***

	estimate	s.e.	t(22)	t pr.
Constant	0.2402	0.0971	2.47	0.022
AgeT	-0.00248	0.00183	-1.36	0.189
cancer_dur	-0.0085	0.0142	-0.60	0.553
change scores 1	0.2151	0.0471	4.56	<.001
drug ATM	-0.2631	0.0735	-3.58	0.002
drug NP	-0.0610	0.0559	-1.09	0.287

```
drug OT                          -0.0713   0.0661   -1.08    0.292
drugUse_dur                       0.0606   0.0284    2.13    0.045

Parameters for factors are differences compared with the
reference level:
Factor Reference level
change scores 0
drug AK

43 RCHECK [RMETHOD=deviance; GRAPHICS=high] residual; normal
  44 "General Model."
45 MODEL [DISTRIBUTION=normal; LINK=identity; DISPERSION=*]


change_abs
46 FIT [PRINT=model, summary, correlations, estimates;
CONSTANT=estimate; FPROB=yes; TPROB=yes; \
  47    FACT=9] +drugUse_dur
```

Model checking

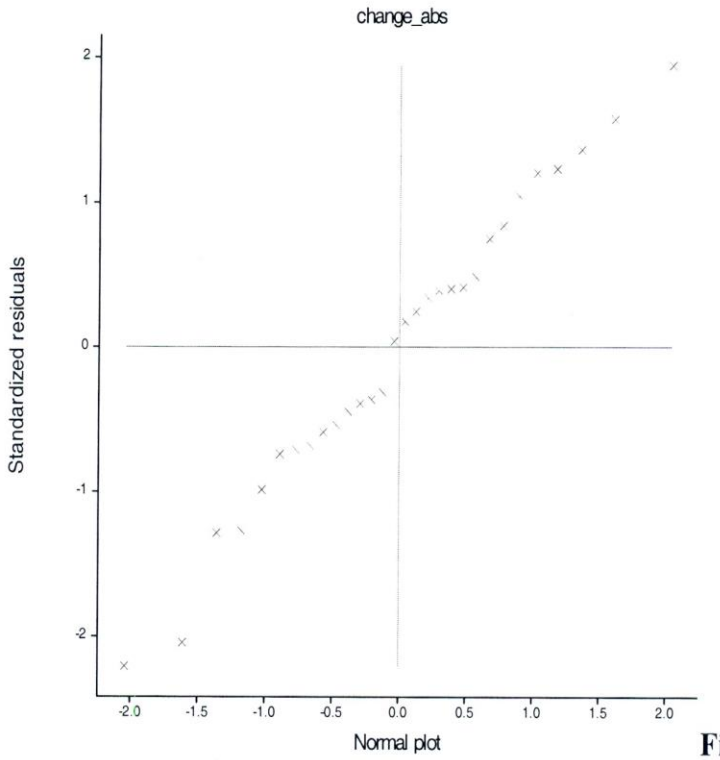


Figure 6: Model checking for all the variables

4.1.4.2 Results - $Y=\beta_0+\beta_1T+\beta_2\Delta+\beta_3V+\beta_4d+\varepsilon$

Cancer duration removed from model (27), leading to

$$Y=\beta_0+\beta_1T+\beta_2\Delta+\beta_3V+\beta_4d+\varepsilon \quad (2)$$

Where y=absolute change

T=patients age

Δ=change scores

V=class of drug used

d=drug use duration

GenStat output 2: Cancer duration removed from genStat output (1)

***** Regression Analysis *****

Response variate: change_abs

Fitted terms: Constant + AgeT + change_scores + drug + drugUse_dur

*** Summary of analysis ***

	d. f.	s.s.	m.s.	v.r.	F pr.
Regression	6	0.7817	0.13028	12.81	<.001
Residual	23	0.2339	0.01017		
Total	29	1.0156	0.03502		

Percentage variance accounted for 71.0

Standard error of observations is estimated to be 0.101

* MESSAGE: The following units have large standardized residuals:

Unit	Response	Residual
9	0.253	-2.15
27	0.056	-2.13

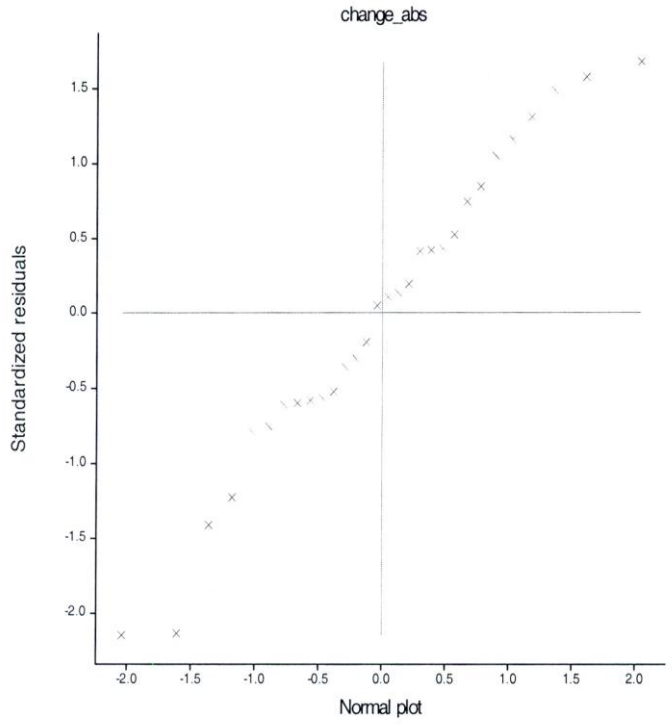
*** Estimates of parameters ***

	estimate	s.e.	t(23)	t pr.
Constant	0.2294	0.0941	2.44	0.023
AgeT	-0.00262	0.00179	-1.46	0.157
change_scores 1	0.2185	0.046	4.73	<.001
drug ATM	-0.2602	0.0723	-3.60	0.002
drug NP	-0.0700	0.0532	-1.32	0.201
drug OT	-0.0748	0.0650	-1.15	0.262
drugUse_dur	0.0504	0.0226	2.23	0.036

```
Parameters for factors are differences compared with
the reference level:
Factor Reference level
  change_scores  0

drug AK
```

Model checking



\FIGURE 7: Model checking where cancer duration is removed.

4.1.4.3 Discussion

The kinds of drug used by patients contribute to the fading fingerprint. Given the major class of drugs used. We classify the data into alkylating, antimetabolic, others, natural product and estimate the probability of change caused by particular class of drug. It is clear that, drug effect on fading fingerprint have a more complex structure and their components are not exactly exchangeable since the individual change probabilities P_1 P_2, Pi where i = 1, 2, ...4 are not identically to probability obtained in the full model.

4.2 STOCHASTIC MODELING OF PROCESSES LEADING TO FADING FINGERPRINTS

We consider a situation where four class active drugs are used by patients. Tumor cells may be in one of four mutually exclusive states defined by sensitivity to the drugs *T*l, *TZ*, *T*3 and *T4: RO* (sensitive to the four class of drugs), *R1* (resistant to Tl and sensitive to T2, T3, T4), *R2* (resistant to *T2* and sensitive to T1, T3, T4) or *R3* (resistant to T3 and sensitive to *T*1, T2, T4) Let *Ri (t)* be the number of cells in the ith compartment at time t. Each compartment is assumed to grow with the kinetics of a pure birth process with compartment specific rates *bi* (*t*)*Ri* (*t*) ,

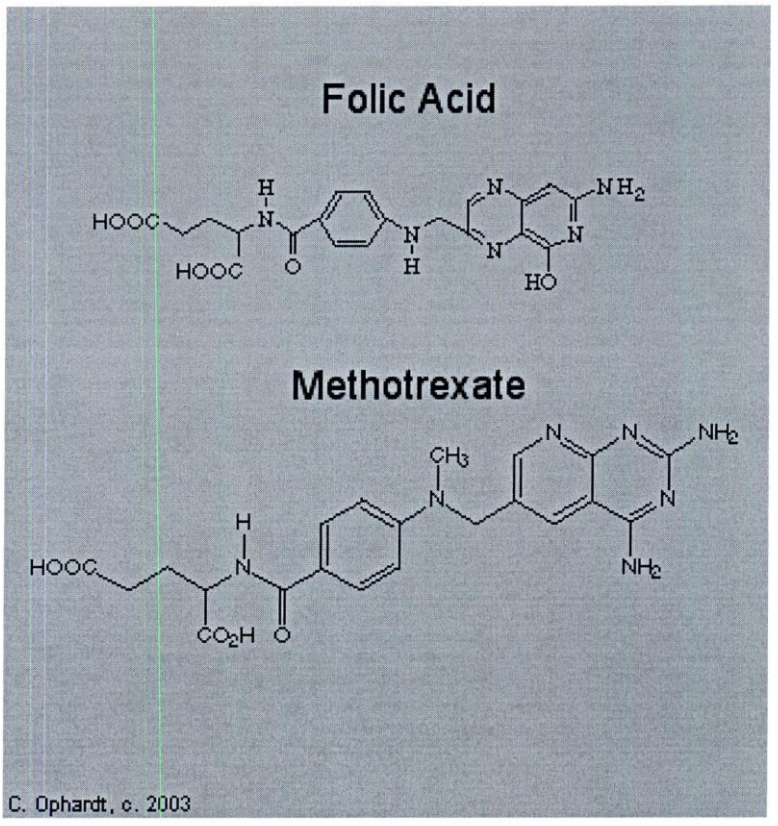
i = 0, 1, 2, 3. Transitions are assumed to occur between compartments with a constant probability per division, a_{ik}, where i is the index of the originator state and k is the destination state. Each tumor cell is assumed to obey the log-kill law in its response to drugs in which the log of the probability of cell survival PD is proportional to the drug dose, i.e., In $(P_D (d_k))$ = In $(P$ {tumor cell survival}) = $-p_{ik} d_k$ *(3)*

where d_k is the dose of drug T_k , k =1, 2 and p_{ik} is the parameter for drug Tk in cells of type i .

In what follows, we will assume we have available treatment times, t_j j =1,...., N on a scale where t = 0 represents the time the tumor developed (1 cell). The probability that the tumor is cured at time t is taken to be equivalent to the probability that there is no tumor cells alive, i.e., $P\{RO(t) = 0, R1(t) = 0, R2(t) = 0, R3(t) = O\} = P\{R(t) = O\} = \Psi R(t)(s)$ is the probability generating function (PGF) of the process $R(t)$ then $P\{R(t) = O\} = \Psi R(t)(0)$ (4)

Thus if we can calculate the PGF we can obtain the required probability by evaluating it at a particular point s = 0 . We can obtain an expression for this PGF by using the well known relationship that if Yi, i =1, 2, ... are independent identically distributed integer valued stochastic processes and N is another independent integer valued process, then the process $Z = \sum_{n=1}^{N} Y_n$ (5)

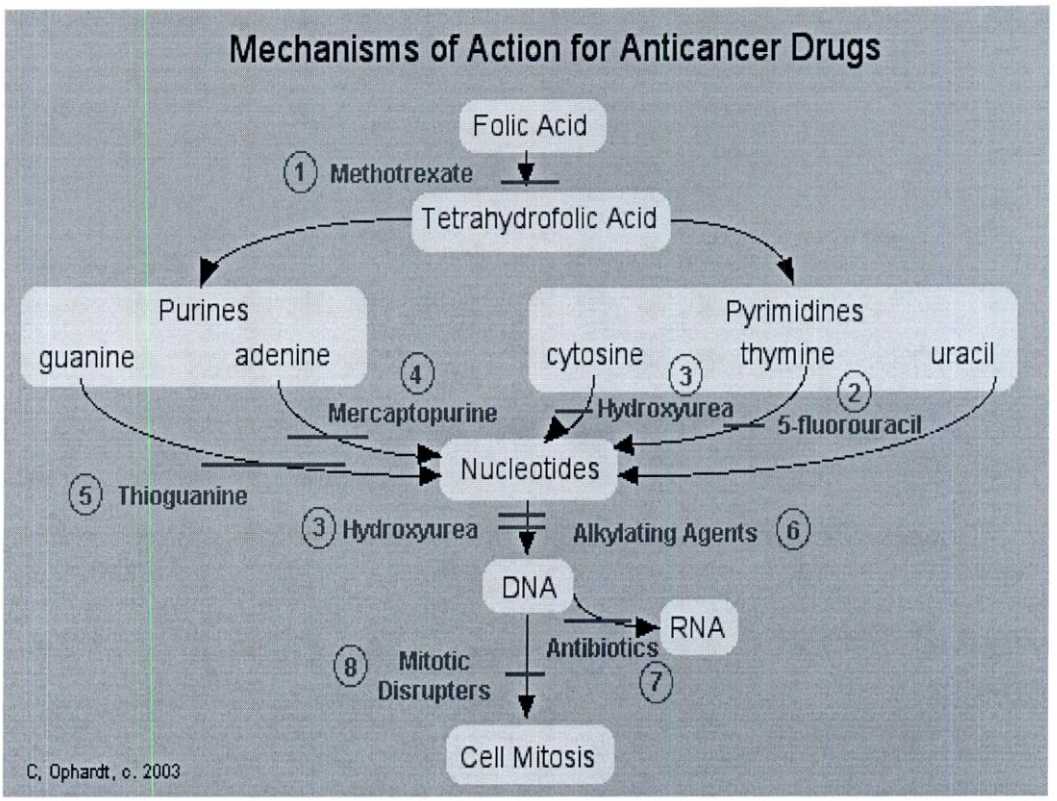
Y_N has PGF given by $\Psi Z(s) = \Psi N(\Psi Y(s))$ where $\Psi N(s)$ and $\Psi Y(s)$ are the PGFs of N and Y. In particular we have that the PGF after treatment at time t, is given by the PGF prior to treatment at time t- evaluated at a point given by the PGF of the effect of treatment on a single cell.



An extract from W. C. Evans (2002) , Pharmaconosy

Figure 2 the chemical structure of folic acid and methotrexate

An example of antimetabolic drug interfering with replication, transcription and DNA synthesis,



An extract from W. C. Evans (2002), Pharmaconosy

Figure 3: pharmacokinetic trajectory of cancer drugs, DNA modification and RNA damage. The drugs initiate hydroxyl radical ($OH^{\cdot}$) which attack and generate a whole series of modified purine and pyrimidine bases.

Stochastic model for toxic accumulation in cells

All the four class of drugs are assumed to have unwanted dose dependent toxic effects on one or more normal systems. This will be summarized in a single variable X which is equal to the logarithm of the size of the critical normal population which is assumed to re-populate following a Gompertzian form of growth, i.e., $x(t) = x_\infty - (x_\infty - x_s)e^{-kit}$ *(6)*

where *s x* is the asymptotic size, k_1 a growth parameter and t is the elapsed time from when the system was of size x_∞ If the normal system is perturbed, then its re-growth is described by the

same equation. The anticancer drugs T_k are assumed to perturb the normal system, indicated by

Δx, following a log-kill law so that $\Delta x = {}^{-} - P_{xk}{}^{dk}$ (7)

In attempting to model clinical cancer the important outcome associated with the effect of chemotherapy on the normal tissue is the occurrence of a toxic event. The toxic event can represent a variety of situations. As well as the most drastic, death of the patient, it can also typify a medical outcome such as kidney failure or neurological damage, that the therapist is trying to avoid. On a more basic level it denotes any outcome that causes the cessation of treatment. A commonly used model for the probability of the toxic (or therapeutic) effect, P_T , of single doses of the drugs is the logistic function, i.e.,

$P_T(d_k) - 1- [1 + e^{\beta 0}][1 + e^{\beta 0 + \beta ik}]^{-1}$ (8)

where β_0 and $\beta > 0$ are constants

We may combine Eqs. (6) and (7) to provide a formula relating changes in the level of the normal system from its physiologic value to the probability of a toxic event, i.e.,

$P_T(\Delta x) = 1 - [1 + e^{\beta 0}][1 + e^{\beta 0 - \beta \Delta}]^{-1}$. (9)

We assume in Eq. (7) that the determinant of the likelihood of toxicity is only influenced by the net kill on the normal system of the drug and not by which drug is used. One of the characteristics of cancer chemotherapy is that normal systems are being repeatedly perturbed by the ongoing sequential application of therapy and may not return to their physiologic values during the course of therapy. We may utilize the parameterization of Eq. (7) to model this situation as follows. If $x(t^{-})$ is the size of the normal system prior to the administration of a drug dose at time t and $x(t)$ is the size after (as given in Eq. (11)), then the probability of a toxic event associated with this dose is given by

$P_T(t) = P_T(x(t) = P_T(x(t) - x_\infty) - P_T(x(t^{-}) - x_\infty)/(1 - P_T(x(t-) - x_\infty))$

$= 1 - (1 - \exp[\beta_0 + \beta(x_\infty - x(t^{-}))]/(1- \exp[\beta_0 + \beta(x_\infty - x(t))])$. (10)

Eq. (8) provides an expression for the probability of toxicity conditional on no preceding toxic event.

Using Bayes theorem we may simply calculate the cumulative probability of a toxic event, CUMPT (t), from $\mathrm{CUMP_T}(tj) - \mathrm{CUMP_T}(t_j{}^{-}) = P_T(t)[1-CUMPT(t_j^{-})]$ (11)

with the condition $CUMPT(t_j-) = 0$. The toxicity expand the normal causing swelling and peeling of the ridges.

If no control is applied, the model is simple and cancer cell grows exponentially. Roughly, sensitive cells grow at a certain rate and this model is constructed on the basis of turnover process of cells, a stochastic process of genetic mutations, cell fitness mutation rates, number of stages and many other characteristics affect the original cell of an individual cell. In this case, for homozygous individuals, two mutations would be required to initiate the process. Basically, the changes causing DNA damage and PCR decay can be presented in the form of a successive of the stages since mutations are fixed during cell replication. It is therefore natural to model gene mutation

on the basis of a process of cell turnover. Such process can be described by the Markov chain. The model can be used to compute the probability of the incidence of cancer on physical mapping of DNA at a particular age ***t***. This will of course be function not only of the age, but also of the number stages in long term trend analysis. Let *Set)* be the survival probability for finger cancer, that is the probability that a randomly chosen finger cell is free from infection up

to age t. On this note the cell hazard rate then satisfies $h(t) = - s:(t) /o(t)$ and inversely (12)

$$S(t) = exp\{- h(t))= = exp\{-f_0^t h(u)du\} \quad (13)$$

and the probability that finger cells die as a result of turnover is

$$p(t) = \lim_{\Delta t \to 0}[pr\{t < x < t + \Delta t I x > t\}/\Delta t] \quad (14)$$

For any proposed model, we can adjust the parameters in such a way that h (t) closely resembles the incidence rates as derived from the observed changes. In this way, it is convincing to note that a single stage model cannot explain the phenomenon of rising genetic mutation that impedes DNA repair mechanism. Parameters in this model have biological meaning and take in values that are in agreement with current knowledge in cancer growth (Tan et al., 1991). In a two stage model, the normal cells undergo initiation stage that produces an irreversible change and leads to a growth advantage for initiated cells. The major protection against cancer may reside in efficiency of DNA repair and successful initiation may require both DNA alteration and some degree of cell proliferation to allow the change to be fixed in the DNA .Such cells can either die or by chance undergo a promotion process which induced the further changes. Dead cells begin when a dead receptor on the doomed cell plasma membrane receives a signal to die, within seconds enzymes caspases are activated inside the cell stimulating each other and snipping apart various cells components. These killer enzymes demolish the enzymes that replicate and repair DNA which affect biological reactions such as glycolysis and TCA cycle in cytoplasm and mitochondria respectively. To model these changes statistically, we refer to Table 4and denote N(t), the number of normal cells at age***t***. *T*(*t*),The number of stem cells at age ***t***.at the beginning of replications

N(0) = *No* and T(o) =T_o, the initial number of cells where *No* = 1.5 x 10^{10} *(biological constant)*, n the number of mutations necessary to produce an initiated cell, *q;* the number of cell division per year.

Let $r_1, r_2, r_3, \ldots r_n$, where r = 7 x 10^{-7} *(biological constant),*the mutation rates per cell division for the n mutation. To show that initiated cells appear in ones organs according to nonhomogeneous Poisson process with a rate depending on the details of the initiation process. The cells in the tissues turn over that is they either divide and double or they simply die. The conceptually simplest method treats the cells as synchronized and turnover at regular intervals. In reality, the cellular alteration and the interactions between cell replication are more complex than what goes into this model, but for the purpose of this model, these complications will not be catered for. Let us treat stem cells as eternally living cells at each step; they divide with probability of one (certain), thereby producing a replacement stem cell and one new normal cell. This normal cell then divides a few times before it eventually dies. At each turning over, half the normal cells die and are replaced again through the doubling of the remaining cells in the unit.

To relate this process to the age in years ***t***, let us define an additional parameter, the number of turnovers per year. It is biologically reasonable to assume τ = 3, that is three turnovers per year. It is therefore convincing to show that if N (t), total number of finger normal cells is very small, and that during a life-span of a human almost more number of divisions takes place.
Mutation becomes fixed in a cell at the time of division. One of the two new cells created by the doubling process may carry a particular mutation. The mutation rate per division is equal to the probability that one of the daughter cells carries the mutation and dies within a few turnovers. These dead cells begin when dead receptor on the doomed cells plasma membrane receives a signal die within seconds and enzymes caspases are activated. Inside the cell, stimulating each other and snipping apart various cell components. These killer enzymes demolish the enzymes that replicate and repair DNA. Caspases also activate enzymes that chew DNA up into pieces and destroy the cell's ability to adhere to other cells.

Let *M(t),* denote the number of stem cells carrying a particular mutation that occurs at rater$\boldsymbol{\tau}_1$, the expected number of mutated stem cells is given as

$$r_1 To + r_1(1- r_1)T_o + \ldots + r_1(1- r_1)^{k-1} T_o \quad (15)$$

$$= To\,[1- (1- r_1)^k = T_0 r_1 k \quad (16)$$

It is mathematically convenient to work with stochastic process in continuous time. This model can be based on the concept of Markov chains in continuous time. We can describe the behavior of M (t) by the rule

$P[M(t + h) = i + M(t) = i] = T(t)uh + o(h)$ (17)

This expresses the probability that at age t $+h$ mutation has been created at the palm knowing very well that at age (t), there are i mutated cells which either survive or die. The term $o(h)$ satisfies $\frac{o(h)}{h} \rightarrow$ o as

$h \rightarrow$ o, the number of happenings in non-overlapping time interval is independent.

Using the Markov property, this rule can be transformed into equations for the probabilities

$P_i = P[M(t) = M(0)] = 0$ (18)

The Kolmogorov forward equation

$P_j(t) = T(t)uP_{j\ -1}$ (t)-T(t)uPj(t) (19)

This implies a differential equation for the generating function $\emptyset(t,s) = \sum_{j\ -0}^{\infty} P_j(t)s^{j(32)}$ is

$\frac{\partial \emptyset}{\partial t} = uT(t)(s- 1)\emptyset(t,s)$ (20)

Solving this equation under the condition that

$\emptyset(t = o, s) = 1$ leads to $\emptyset(t, s) = \exp [u\int_o^t S(u)du(s -1)]$ (21)

That is M (t) has a Poisson distribution with mean $u\int_o^t S(u)du$. (Expected number of cells carrying mutation). If we substitute $u = r_1q$ we obtain the same result as in (1) which gives a mean of $T_{O}r_1qt$, this is a mean step function that steps upward whenever a new mutated finger cell appears which is different from the original cell.

We can think this process as a homogeneous Poisson process with intensity

$\omega = To\tau_1q$ (22)

If a second mutation were required for initiation and it is introduced in the continuous time model at rate r_2q, the rate for the creation of cells having first mutation and subsequently the

second one would be approximately equal to $r\tau_2q\int_o^t To\ r_2qdu = r_1r_2qT_oq^2t$ (23)

Equation (39) is explained by the fact that all the cells having the first mutation being created in the interval from 0 to t can mutate at t into a cell carrying both mutations.

It should be noted that, the details of the computation depend to some extent on whether this second mutation is at the same clonal site on the second strand or the whether it is unrelated. Second mutation at the same site motivates strand breakage in PCR and distorts transcription in protein synthesis. The process of transcription cannot copy a particular part of the DNA sequence of a chromosome into an RNA molecule that is one strand of the DNA double helix. Generalizing the above approach shows that the double mutant finger cells can be modeled as arising from non-homogeneous Poisson process with mutation intensity function

$\omega_1(t) = 2r_{1r2},Noq^2$ (24)

A further generalization of these formulas allows us to take fluctuations in finger pad ridges, in

this case $\omega_1(t) = 2r_1r_2,N(t)q^2t$ (25)

and the number of double mutated PCR at age t follows a Poisson distribution with expectation

$\int_0^t \omega_1(\mu)\text{du} = 2\ \omega_1(t) = 2\tau_{1r2}q^2 \int_o^t N(u)udu$ (26)

In general, for n mutations, the rate function has biological constants $r_{1..}r_n\text{N(t)}q^n$. In this case

the function that depends only on the number of mutation required for initiation $n = \int(n)t^{n-l}$ *the*

intensity function of this Poisson process is intimately related with the hazard rate for acquiring the mutations. This follows from the particular form of the survival function in this special case,

which is equal to $(t) = P[l(t) = 0] = \exp[-\Delta(t)] = \exp\ [-\int_o^t \omega_1\ (\mu)du]$ (27)

It follows from the above model that the hazard rate is $h_1(t) = 2\tau_1\tau_2N(t)q^2\text{t}$ (28)

TABLE 4: BIOLOGICAL CONSTANTS

Birth of cell (β)	death of cell (δ)	Mutation rate per cell division (r)	Number of cells at time *t*
9	8.9	7×10^{-7}	$1.5\times$

10^{10}

4.2.1 Clonal expansion

We want to analyze the effect of cancer beyond second mutation.

In the model, we defined C (t), the number of cells in a clonal expansion due to the growth of an initiated cells created at age a β, the birth rate in the stochastic process controlling the initiated cells. δ, the death rate of the process controlling the initiated cells.

The mutation at age ***a*** gives rise to a subsequent clonal expansion. The simplest model for such an expansion is a birth and death process with a bigger death rate than the birth rate. This is a Markov chain with the state $C(t)$ being the number of cells in the clone at time $t > a$. To keep things simple, we explain that each cell in the clone divides with birth rate β and disappears with death rate δ and act independently of the other cells. In the birth case an additional cell appears which means that a transition from $C(t)$ *to* $C(t) + 1$ occurs. Our interest here lies in the death case and it is equally useful in the model $C(t)$ changes to $C(t) - 1$. The probability for the death transition is

$$[C(t+h) = C(t) - 1/C(t) = c] = c\delta h + 0(h) \; at \; \delta > \beta \qquad (29)$$

The expected number of cells in the clone dies exponentially $E[C(t) = \exp(\delta - \beta)(t - \alpha)]$ for $t \geq a$ .When $\delta = \beta$ the colony ridges is sure to break and disappear after a finite time t whereas, for $\delta > \beta$, the colony die and eliminated to age $t = \infty$ with probability $(\delta - \beta)/\delta$. A

dying colony in this case is eliminated nearly in $E[C(t)/C(t) > 0] = \frac{\delta}{\delta - \beta}$, (30)

at this stage, the pseudo genes are also found at specific mutation points and they are comparable with many olfactory receptors coding. The so-called pseudo genes which form as a result of frame shift mutation as well as mutations that result in premature stop codon and lose their functionalities.

Roughly, sensitive cells grow at the rate βs and resistant cells grow at rate $\beta r 0$, or, more

precisely; $R + S = \beta s S + \beta r R.$ (31)

The quotient $x = \frac{s}{R}$ satisfies a Riccati equation of the form

$$x = r\beta_R + (1- q)\beta s - (1- r)\beta_R) x - q\beta s x^2 \quad (32)$$

with positive initial condition x (0) > O. Clearly the interval [0,1) is invariant for (48) and this equation has a unique stable equilibrium in this interval at

$$x = \frac{(1 - q)(\beta s-(1-r)\beta r+\sqrt{((1- q)\beta s - (1-r)\beta^2+4rq\beta s\beta r}}{2r\beta_r} > 0 \quad (33)$$

Thus, left alone, cancer cells grow exponentially reaching the relative proportions S = ẍR.

5.0 CONCLUSION AND RECOMMENDATIONS

The reliability of the model is assessed using a criteria based on the degree to which the models are able to capture the observed changes in the fingerprint images. In fact, forensic experts and most automatic fingerprint matching systems use minutiae for identification since these features have been shown to be stable and can be reliably extracted from points. There are many types of ridge anomalies that occur in fingerprint image, these include ridge endings, bifurcations, islands, dots, enclosures, bridges, double bifurcations, trifurcations and many others. However, in this work, consideration has been given to ridge fading The main reason for this is that, the occurrence of transitional fading of the other ridge anomalies is relatively rare, and it is easy to constantly detect sharp fading rates in ridges compare to other minutiae types.

We have presented a general statistical framework, along with several stochastic models for fading in fingerprint, from which the distribution of change scores and the extreme value distributions of the families of cancer drugs contributing to the change in patients fingerprint can be accurately predicted.

In section 2 on stochastic modeling, we have shown the age-dependent risk of a theoretical model of effect of cancer on DNA and PCR damage incorporating the familiar notions of multiple stages. It is however assumed that mutation which leads to cell turnover causes growth advantage where cells either die or proliferate. This breaks DNA strands and affects bonds in codons of PCR. Under these conditions, the intensity of cancer and the hazard of acquiring depleted cells can be calculated. We use biologically reasonable constants of $N(t) = 1.5 \times 10^{10}$ (the total number of cells in the body). $\tau_1 = \tau_2 = \tau_3 = 0.7 \times 10^{-7}$ .q = 3(The number of turnovers per year).

Once a cell is initiated with mutation, we expect β = 9.0 (the rate of birth of new cells) and δ = 8.9 (the rate of death of old cells).

As shown in the model (genStat output 2), there is a probability of change (fading) of 0.0504 as patients use cancer drugs every six months. Ageing did not cause a change in fingerprint. Drug AK cause greater changed in fingerprint than ATM by 26%, this is significant at 0.05 α - level. There was no substantial difference between drug AK and OT, NP in causing changes in fingerprint. The change depended more on the duration of drug use; the longer the duration, the more the change (fade).On the basis the results from the study, we strongly recommend that, agencies like Biometric Passport Officials, e-zwich, Security Officials such as Bureau of National Investigation (BNI), Criminal Investigation Department (CID) and Court of Law should not solely depend on fingerprint for personal identification, other (biometric) measures of personal identification should be used alongside with fingerprint. It is also recommended that e-zwich machines should make an allowance of 0.0504 (+ standard error) for known cancer patients on cancer drugs to ensure uninterrupted access to their accounts. Furthermore, a mechanism must be put in place that allows known cancer patients to renew their Biometric Passport regularly.

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APPENDIX

```
***** Regression Analysis *****

Response variate: change_abs
     Fitted terms: Constant + AgeT + cancer_dur + change_scores
                   + drug + drugUse_dur


*** Summary of analysis ***

              d.f.          s.s.          m.s.       v.r.  F  pr.
Regression       7        0.7855       0.11221       10.73 <.001
Residual        22        0.2301       0.01046
Total           29        1.0156       0.03502

Percentage variance accounted for 70.1
Standard error of observations is estimated to be 0.102
* MESSAGE: The following units have large standardized
residuals:
         unit      Response     Residual -
            9         0.253         -2.04
           27         0.056        -2.21


*** Estimates   of parameters  ***

                                 estimate          s.e.      t(22)   t pr.
Constant                           0.2402        0.0971       2.47    0.022
AgeT                              -0.00248       0.00183     -1. 36   0.189
cancer_dur                         -0.0085        0.0142      -0.60    0.553
change_scores    1                  0.2151        0.0471       4.56    <.001
drug ATM                           -0.2631        0.0735      -3.58    0.002
drug NP                            -0.0610        0.0559      -1.09    0.287
drug OT                            -0.0713        0.0661      -1.08    0.292
drugUse_dur                         0.0606        0.0284       2.13    0.045

Parameters  for  factors  are  differences  compared with  the reference    level:
             Factor Reference level
       change_scores 0
                drug AK
                                    41
 43 RCHECK [RMETHOD=deviance; GRAPHICS=high] residual; normal
 44 "General Model."
 45 MODEL [DISTRIBUTION=normal; LINK=identity; DISPERSION=*]
 change_abs
 46     FIT [PRINT=model,summary,correlations,estimates;
CONSTANT=estimate; FPROB=yes; TPROB=yes;\
```

47 FACT= 9] +drugUse_dur

```
47.....................................................................
...


***** Regression Analysis *****

 Response variate: change_abs
     Fitted terms: Constant, drugUse_dur


*** Summary of analysis ***

              d.f.         s.s.         m.s.      v.r.  F pr.
Regression       1        0.005      0.00469      0.13  0.721
Residual        28        1.011      0.03610
Total           29        1.016      0.03502

Residual variance exceeds variance of response variate
Standard error of observations is estimated to be 0.190
* MESSAGE: The following units have high leverage:
         Unit     Response    Leverage
           13        0.112       0.207
           16        0.074       0.164
           18        0.427       0.361


*** Estimates of parameters ***

                 estimate         s.e.     t(28)  t pr.
Constant            0.277        0.104      2.66  0.013
drugUse_dur        0.0107       0.0296      0.36  0.721


*** Correlations between parameter estimates ***

estimate      ref    correlations

Constant        1    1.000
drugUse_dur     2   -0.943  1.000
                        1      2

  48  "General Model."
  49  MODEL [DISTRIBUTION=normal; LINK=identity; DISPERSION=*] change_abs
  50    FIT  [PRINT=model,summary,correlations,estimates;  CONSTANT=estimate;
FPROB=yes; TPROB=yes;\
  51   FACT=9] +drugUse_dur
```

```
51.....................................................................
...


***** Regression Analysis *****

 Response variate: change_abs
     Fitted terms: Constant, drugUse_dur


*** Summary of analysis ***

             d.f.         s.s.         m.s.      v.r.  F pr.
Regression      1        0.005      0.00469      0.13  0.721
Residual       28        1.011      0.03610
Total          29        1.016      0.03502

Residual variance exceeds variance of response variate
Standard error of observations is estimated to be 0.190
* MESSAGE: The following units have high leverage:
         Unit     Response    Leverage
           13        0.112       0.207
           16        0.074       0.164
           18        0.427       0.361


*** Estimates of parameters ***

                 estimate         s.e.     t(28)  t pr.
Constant            0.277        0.104      2.66  0.013
drugUse_dur        0.0107       0.0296      0.36  0.721


*** Correlations between parameter estimates ***

estimate      ref    correlations

Constant        1    1.000
drugUse_dur     2   -0.943  1.000
                        1      2

  52  "General Model."
  53  MODEL [DISTRIBUTION=normal; LINK=identity; DISPERSION=*] change_abs
  54    FIT  [PRINT=model,summary,correlations,estimates;  CONSTANT=estimate;
FPROB=yes; TPROB=yes;\
  55   FACT=9] +drugUse_dur
```


```
55.....................................................................
...

***** Regression Analysis *****

 Response variate: change_abs
     Fitted terms: Constant, drugUse_dur


*** Summary of analysis ***

              d.f.         s.s.         m.s.      v.r.  F pr.
Regression       1        0.005      0.00469      0.13  0.721
Residual        28        1.011      0.03610
Total           29        1.016      0.03502

Residual variance exceeds variance of response variate
Standard error of observations is estimated to be 0.190
* MESSAGE: The following units have high leverage:
         Unit     Response    Leverage
           13        0.112       0.207
           16        0.074       0.164
           18        0.427       0.361


*** Estimates of parameters ***

                 estimate         s.e.     t(28)  t pr.
Constant            0.277        0.104      2.66  0.013
drugUse_dur        0.0107       0.0296      0.36  0.721


*** Correlations between parameter estimates ***

estimate      ref    correlations

Constant        1    1.000
drugUse_dur     2   -0.943  1.000
                        1      2

  56  "General Model."
  57  MODEL [DISTRIBUTION=normal; LINK=identity; DISPERSION=*] change_abs
  58    FIT  [PRINT=model,summary,correlations,estimates;  CONSTANT=estimate;
FPROB=yes; TPROB=yes;\
  59   FACT=9] +drugUse_dur
```

```
59.....................................................................
...


***** Regression Analysis *****

 Response variate: change_abs
     Fitted terms: Constant, drugUse_dur


*** Summary of analysis ***

              d.f.         s.s.         m.s.      v.r.  F pr.
Regression       1        0.005      0.00469      0.13  0.721
Residual        28        1.011      0.03610
Total           29        1.016      0.03502

Residual variance exceeds variance of response variate
Standard error of observations is estimated to be 0.190
* MESSAGE: The following units have high leverage:
         Unit     Response    Leverage
           13        0.112       0.207
           16        0.074       0.164
           18        0.427       0.361


*** Estimates of parameters ***

                 estimate         s.e.     t(28)  t pr.
Constant            0.277        0.104      2.66  0.013
drugUse_dur        0.0107       0.0296      0.36  0.721


*** Correlations between parameter estimates ***

estimate      ref    correlations

Constant        1    1.000
drugUse_dur     2   -0.943  1.000
                        1      2

  60  "General Model."
  61  MODEL [DISTRIBUTION=normal; LINK=identity; DISPERSION=*] change_abs
  62    FIT  [PRINT=model,summary,correlations,estimates;  CONSTANT=estimate;
FPROB=yes; TPROB=yes;\
  63   FACT=9] +drugUse_dur
```

```
63.....................................................................
...


***** Regression Analysis *****

 Response variate: change_abs
     Fitted terms: Constant, drugUse_dur


*** Summary of analysis ***

              d.f.         s.s.         m.s.      v.r.  F pr.
Regression       1        0.005      0.00469      0.13  0.721
Residual        28        1.011      0.03610
Total           29        1.016      0.03502

Residual variance exceeds variance of response variate
Standard error of observations is estimated to be 0.190
* MESSAGE: The following units have high leverage:
         Unit     Response    Leverage
           13        0.112       0.207
           16        0.074       0.164
           18        0.427       0.361


*** Estimates of parameters ***

                  estimate         s.e.     t(28)  t pr.
Constant             0.277        0.104      2.66  0.013
drugUse_dur         0.0107       0.0296      0.36  0.721


*** Correlations between parameter estimates ***

estimate      ref    correlations

Constant        1    1.000
drugUse_dur     2   -0.943  1.000
                        1      2

  64  "General Model."
  65  MODEL [DISTRIBUTION=normal; LINK=identity; DISPERSION=*] change_abs
  66    FIT  [PRINT=model,summary,correlations,estimates;  CONSTANT=estimate;
FPROB=yes; TPROB=yes;\
  67   FACT=9] +drugUse_dur
```

```
67.....................................................................
...


***** Regression Analysis *****

 Response variate: change_abs
     Fitted terms: Constant, drugUse_dur


*** Summary of analysis ***

              d.f.         s.s.         m.s.      v.r.  F pr.
Regression       1        0.005      0.00469      0.13  0.721
Residual        28        1.011      0.03610
Total           29        1.016      0.03502

Residual variance exceeds variance of response variate
Standard error of observations is estimated to be 0.190
* MESSAGE: The following units have high leverage:
         Unit     Response    Leverage
           13        0.112       0.207
           16        0.074       0.164
           18        0.427       0.361


*** Estimates of parameters ***

                  estimate         s.e.     t(28)  t pr.
Constant             0.277        0.104      2.66  0.013
drugUse_dur         0.0107       0.0296      0.36  0.721


*** Correlations between parameter estimates ***

estimate      ref    correlations

Constant        1    1.000
drugUse_dur     2   -0.943  1.000
                        1      2

  68  "General Model."
  69  MODEL [DISTRIBUTION=normal; LINK=identity; DISPERSION=*] change_abs
  70    FIT  [PRINT=model,summary,correlations,estimates;  CONSTANT=estimate;
FPROB=yes; TPROB=yes;\
  71   FACT=9] +drugUse_dur
```

```
71.....................................................................
...


***** Regression Analysis *****

 Response variate: change_abs
     Fitted terms: Constant, drugUse_dur


*** Summary of analysis ***

             d.f.         s.s.         m.s.      v.r.  F pr.
Regression      1        0.005      0.00469      0.13  0.721
Residual       28        1.011      0.03610
Total          29        1.016      0.03502

Residual variance exceeds variance of response variate
Standard error of observations is estimated to be 0.190
* MESSAGE: The following units have high leverage:
         Unit     Response    Leverage
           13        0.112       0.207
           16        0.074       0.164
           18        0.427       0.361


*** Estimates of parameters ***

                 estimate         s.e.     t(28)  t pr.
Constant            0.277        0.104      2.66  0.013
drugUse_dur        0.0107       0.0296      0.36  0.721


*** Correlations between parameter estimates ***

estimate      ref    correlations

Constant        1    1.000
drugUse_dur     2   -0.943  1.000
                        1      2

  72  "General Model."
  73  MODEL [DISTRIBUTION=normal; LINK=identity; DISPERSION=*] change_abs
  74    FIT  [PRINT=model,summary,correlations,estimates;  CONSTANT=estimate;
FPROB=yes; TPROB=yes;\
  75   FACT=9] AgeT+change_scores+drug+drugUse_dur
```


75……

***** Regression Analysis *****

Response variate: change_abs
Fitted terms : Constant + AgeT + change_scores + drug + drugUse_dur

*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.	F pr.
Regression	6	0.7817	0.13028	12.81	<.001
Residual	23	0.2339	0.01017		
Total	29	1.0156	0.03502		

Percentage variance accounted for 71.0
Standard error of observations is estimated to be 0.101
* MESSAGE: The following units have large standardized residuals:

Unit:	Response	Residual
9	0.253	-2.15
27	0.056	-2.13

*** Estimates of parameters ***

		estimate	s.e.	t(23)	t pr.
Constant		0.2294	0.0941	2.44	0.023
AgeT		-0.00262	0.00179	-1.46	0.157
change_scores	1	0.2185	0.0461	4.73	<.001
drug ATM		-0.2602	0.0723	-3.60	0.002
drug NP		-0.0700	0.0532	-1.32	0.201
drug OT		-0.0748	0.0650	-1.15	0.262
drugUse_dur		0.0504	0.0226	2.23	0.036

Parameters for factors are differences compared with the reference level:

Factor	Reference
changes_scores	0
drug	AK

*** Estimates of parameters ***

		estimate	s.e.	t(22)	t pro
Constant		0.2402	0.0971	2.47	0.022
AgeT		-0.00248	0.00183	-1.36	0.189
cancer_dur		-0.0085	0.0142	-0.60	0.553
change_scores	1	0.2151	0.0471	4.56	<.001
drug ATM		-0.2631	0.0735	-3.58	0.002
drug NP		-0.0610	0.0559	-1.09	0.287
drug OT		-0.0713	0.0661	-1. 08	0.292
drugUse_dur		0.0606	0.0284	2.13	0.045

50
*** Correlations between parameter estimates ***

```
estimate                        ref    correlations

Constant                          1    1.000
AgeT                              2   -0.618    1.000
change_scores  1                  3   -0.416    0.243  1.000
drug ATM                          4   -0.068    0.001  0.440  1.000
drug NP                           5    -0.316 -0.147  0.277  0.504  1.000
drug OT                           6    -0.016 -0.468  0.181  0.457  0.586  1.000
drugUse_dur                          7      -0.173 -0.445 -0.378 -0.500 0.014 0.102
1.000
                                            1       2       3       4       5         6
7

   76 "General Model."
   77 MODEL [DISTRIBUTION=normal; LINK=identity; DISPERSION=*] change_abs
   78    FIT    [PRINT=model, summary, correlations, estimates;
         CONSTANT=estimate;
FPROB=yes ; TPROB=yes;\
   79 FACT=9] change_scores+drug+drugUse_dur
```


***** Regression Analysis *****

Response variate: change_abs
Fitted terms: Constant + change_scores + drug + drugUse_dur

*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.	F pr.
Regression	5	0.7599	0.15199	14.27	<.001
Residual	24	0.2556	0.01065		
Total	29	1.0156	0.03502		

Percentage variance accounted for 69.6
Standard error of observations is estimated to be 0.103
* MESSAGE: The following units have large standardized residuals:

Unit	Response	Residual
27	0.056	-2.08

large responses are more variable than small responses

*** Estimates of parameters***

		estimate	s.e.	t(24)	t
Constant		0.1444	0.0757	1.91	0.069
change_scores	1	0.2349	0.0458	5.13	<
drug ATM		-0.2601	0.0740	-3.51	0.002
drug NP		-0.0814	0.0538	-1.51	0.144
drug OT		-0.1193	0.0588	-2.03	0.054
drugUse_dur		0.0357	0.0207	1.73	0.097

Parameters for factors are differences compared with the reference level:

Factor	Reference level
change_scores	0
drug	AK

*** Correlations between parameter estimates ***

estimate	ref	correlations					
Constant	1	1.000					
change_scores	1	2	-0.349	1.000			
drug ATM		3	-0.086	0.453	1.000		
drug NP		4	-0.522	0.326	0.509	1.000	
drug OT		5	-0.439	0.344	0.518	0.591	1.000
drugUse_dur		6	-0.636	-0.310	-0.558	-0.058	-0.134
			1	2	3	4	5

```
80 "General Model."
   81 MODEL [DISTRIBUTION=normal; LINK=identity; DISPERSION=*] change_abs
   82 FIT [PRINT=model,summary,correlations,estimates; CONSTANT=estimate;
FPROB=yes; TPROB=yes;\
   83 FACT=9] change_scores+drug+drugUse_dur
```


***** Regression Analysis *****

Response variate: change_abs
Fitted terms: Constant + change_scores + drug + drugUse_dur

*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.	F pr.
Regression	5	0.7599	0.15199	14.27	<.001
Residual	24	0.2556	0.01065		
Total	29	1.0156	0.03502		

Percentage variance accounted for 69.6
Standard error of observations is estimated to be 0.103
* MESSAGE: The following units have large standardized residuals:

Unit	Response	Residual
27	0.056	-2.08

* MESSAGE: The error variance does not appear to be constant:
large responses are more variable than small
responses

*** Estimates of parameters ***

	estimate	s.e.	t(24)	t pr.
	0.1444	0.0757	1.91	0.069
Constant	0.2349 -	0.0458	5.13	<.001
change_scores 1	0.2601 -	0.0740	-3.51	0.002
drug ATM drug NP	0.0814 -	0.0538	-1.51	0.144
drug OT	0.1193	0.0588	-2.03	0.054
drugUse_dur	0.0357	0.0207	1.73	0.097

Parameters for factors are differences compared with the reference level:

Factor	Reference level
Change_scores	0
drug	AK

*** Correlations between parameter estimates ***

estimate		ref	correlations					
Constant		1	1.000					
change_scores	1	2	-0.349	1.000				
drug ATM		3	-0.086	0.453	1.000			
drug NP		4	-0.522	0.326	0.509	1.000		
drug OT		5	-0.439	0.344	0.518	0.591	1.000	
drugUse_dur		6	-0.636	-0.310	-0.558	-0.058	-0.134	1.000
			1	2	3	4	5	6

```
  84 RCHECK [RMETHOD=deviance; GRAPHICS=high] residual; normal
  85 "General Model."
  86 MODEL [DISTRIBUTION=normalj LINK=squarerootj DISPERSION=*] change_abs
  87 FIT [PRINT=model,summary,correlations,estimates; CONSTANT=estimate;
FPROB=yes; TPROB=yes;\
  88 FACT=9] change_scores+drug+drugUse_dur
```


***** Regression Analysis *****

Response variate: change_abs
Link function: Square root
Fitted terms: Constant + change_scores + drug + drugUse_dur

*** Summary of analysis ***

	d.t.	s.s.	m.s.	v.r.	F pro
Regression	5	0.7670	0.15341	14.81	<.001
Residual	24	0.2485	0.01035		
Total	29	1.0156	0.03502		

Percentage variance accounted for 70.4
Standard error of observations is estimated to be 0.102
* MESSAGE: The following units have large standardized residuals:

Unit	Response	Residual
9	0.253	-2.14
27	0.056	-2.05

* MESSAGE: The error variance does not appear to be constant:
large responses are more variable than small responses
* MESSAGE: The following units have high leverage:

Unit	Response	Leverage
18	0.427	0.56

*** Estimates of parameters ***

	estimate	s.e.	t(24)	t pr.
	0.3529	0.0791	4.46	<.001
Constant	-0.2638	0.0567	4.65	<.001
change_scores 1	-0.2309	0.0742	-3.11	0.005
drug ATM drug NP	-0.0537	0.0416	-1.29	0.209
drug OT	-0.0833	0.0477	-1.75	0.093
drugUse_dur	0.0268	0.0215	1.24	0.225

Parameters for factors are differences compared with the reference level:

Factor	Reference level
change_scores	0
drug	AK

*** Correlations between parameter estimates ***

estimate	ref	correlations

Constant		1	1.000				
change_scores	1	2	-0.495	1.000			
drug ATM		3	0.155	0.297	1.000		
drug NP		4	-0.270	0.253	0.380	1.000	
drug OT		5	-0.265	0.249	0.329	0.454	1.000
drugUse_dur		6	-0.634	-0.268	-0.602	-0.172	-0.139
			1	2	3	4	5

```
  89   "Gener'alModel."
  90 MODEL [DISTRIBUTION=normal; LINK=probit; DISPERSION=*] change_abs
  91 FIT [PRINT=model,summary,correlations,estimates; CONSTANT=estimate;
FPROB=yes; TPROB=yes;\
  92    FACT=:9] change_scores+drug+drugUse_dur
```


***** Regression Analysis *****

Fitted terms: Constant + change_scores + drug + drugUse_dur

*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.	F pr.
Regression	5	0.7704	0.15407	15.08	<.001
Residual	24	0.2452	0.01022		
Total	29	1.0156	0.03502		

Percentage variance accounted for 70.8
Standard error of observations is estimated to be 0.101
* MESSAGE: The following units have large standardized residuals:

Unit	Response	Residual
9	0.253	-2.18

* MESSAGE: The error variance does not appear to be constant:
large responses are more variable than small responses *
MESSAGE: The following units have high leverage:

Unit	Response	Leverage
18	0.427	0.61
27	0.056	0.42

*** Estimates of parameters ***

	estimate	s.e.	t(24)	t pr.
Constant	-1.168	0.270	-4.32	<.001
change_scores	-0.862	0.195	4.41	<.001
1 drug ATM	-0.771	0.245	-3.14	0.004
drug NP	-0.178	0.141	-1.26	0.221
drugOT	0.276	0.159	-1.74	0.095
drugUse_dur	0.0903	0.0713	1.27	0.217

Parameters for factors are differences compared with the reference level:

Factor	Reference level
changes_scores	0
drug	AK

```
*** Correlations   between  parameter   estimates  ***

estimate                    ref       correlations

Constant                        1     1.000
change_scores   1               2    -0.527   1.000
drug ATM                        3     0.159   0.254   1.000
drug NP                         4    -0.276   0.242   0.394   1.000
drug OT                         5    -0.270   0.232   0.343   0.476   1.000
drugUse_dur                     6    -0.618  -0.246  -0.605  -0.176  -0.140   1.000
                                         1       2       3       4       5       6

   93 "General Model."
   94 MODEL [DISTRIBUTION=normal; LINK=probit; DISPERSION=*] change_abs
   95    FIT    [PRINT=model,summary,correlations,estimatesi    CONSTANT=estimate;
FPROB=yes; TPROB=yes;\
   96    FACT=:9] change_scores+drug+drugUse_dur
```


96.....
...

***** Regression Analysis *****

Response variate: change_abs
Link function: Probit
Fitted terms: Constant + change_scores + drug + drugUse_dur

*** Summary of analysis ***

	d.f.	s.s.	m.s.	v.r.	F pr.
Regression	5	0.7704	0.15407	15.08	<.001
Residual	24	0.2452	0.01022		
Total	29	1.0156	0.03502		

Percentage variance accounted for 70.8
Standard error of observations is estimated to be 0.101
* MESSAGE: The following units have large standardized residuals:

Unit	Response	Residual
9	0.253	-2.18

* MESSAGE: The error variance does not appear to be constant:
large responses are more variable than small responses
* MESSAGE: The following units have high leverage:

Unit	Response	Leverage
18	0.427	0.61
27	0.056	0.42

*** Estimates of parameters ***

	estimate	s.e.	t(24)	t pr.
Constant	-1.168	0.270	-4.32	<.001
change_scores 1	0.862	0.195	4.41	<.001
drug ATM	-0.771	0.245	-3.14	0.004
drug NP	-0.178	0.141	-1.26	0.221
drug OT	-0.276	0.159	-1.74	0.095
drugUse_dur	0.0903	0.0713	1.27	0.217

Parameters for factors are differences compared with the reference level:

Factor	Reference level
change_scores	0
drug	AK

*** Correlations between parameter estimates ***

estimate	ref	correlations				
Constant	1	1.000				
change_scores 1	2	-0.527	1.000			
drug ATM	3	0.159	0.254	1.000		
drug NP	4	-0.276	0.242	0.394	1.000	
drug OT	5	-0.270	0.232	0.343	0.476	1.000
drugUse_dur	6	-0.618	-0.246	-0.605	-0.176	-0.140	1.000