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Liver Material Pathological Sample Identification for Timeline Establishment in the Algor Mortis Process

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Abstract. This contribution presents a mathematical model for the identification pathological samples of liver mass which affect the temperature values in the cooling process of biological material. Pathologic clostridium involves approx. 100 species which include common free-living bacteria as well as other significant pathogens. They especially involve: C. botulinum, C. difficile, C. perfringens, C. tetani, Salmonella, Campylobacter, and Haemophilus bacteria. Research was focused on the Aerococcus viridans type of bacteria strains, wherein the bacteria are sometimes encountered as human pathogens - especially in bacterial endocarditis, and further, Micrococcus Luteus Saprothopic bacteria belonging to the genus Micrococcaceae, as well as Staphylococcus xylosus, Pasteurella pneumotropica, and Staphylococcus nepalensis.

Introduction

Theories of aging are many and not one of them can, with certainty, attempt to explain this process in its full complexity. It is, of course, clear that the maximum age of an individual is genetically determined and that evolution was directed to the preservation of the species, and not the maximization of an individual´s age. Contemporary scientific studies in this field indicate the maximum attainable age of a person as 115 years.
Experimentally, it has been discovered that human cells have a limited maximum number of divisions. This regulation of growth restriction involves DNA methylation and the length of the end segments of chromosomes – i.e. telomeres. At the cellular DNA damage level, we can imagine the generic term of "damage" as the progressive loss of cells that are no longer able to proliferate. Somatic mutations can lead to the creation of defective proteins, or – as the case may be, may affect cell division and differentiation and thus become the cause of tumor growth.

A further factor is also the problem that the human organism is not set-up for an indefinite survival period, since there is a lack of mechanisms that would eliminate certain waste products from the body. In multicellular organisms, “immortal” gametes remain in some way, which give rise to another organism. Aging and death are therefore, processes that are inherent in every living organism and which allow the survival and further development of the species.

The characteristics and specifics of the measured area

Death is a process of irreversible changes that leads to the arrest of functions characteristic for living systems. Its essence is, above all, the irreversible arrest of metabolism and energy transfers with the external environment. In the course of the death of an organism, all cells rarely die concurrently. Death is usually caused by the interruption of ne of the basic primary systems, without which life is impossible (the atria or gates of death). The liver is located in the upper right

abdominal quadrant in the peritoneal cavity and is oriented in the horizontal orientalis direction and is basically divided into two main lobes. Hepatocytes have enormous metabolisation capacity, utilised in the transformation of substances derived from blood. Blood is delivered to the liver by two main streams, and these are the arterial and venous blood systems. The Hepatic Artery diverges from the abdominal aorta and feeds about one third of the blood supply into the liver, the remainder comes through the Portal Vein, which delivers blood rich in digested nutrients from the intestines and stomach directly to the liver before these substances even reach the systemic circulation. After passing through the liver, the intermingled blood from the Hepatic Artery and Portal Vein is fed into the Inferior Vena Cava. On the microscopic level, the liver has a lobular structure which is made up of cubical hepatocyte cells and the sinusoidal lining that converge in a central lobe. The mixed blood of Hepatic Artery and Portal Vein Sine wave passes through the Sinusoid. The wall the Sinusoid is, compared to other permeable capillaries, more permeable and facilitates the exchange of substances between the blood and hepatocytes. Bile from the liver is drained away by a system of canals which start as tiny channels between two adjacent hepatocytes and drains the bile through the Bile Duct into the duodenum. The bile duct converges in the duodenum together with the pancreatic by-products in the Vater papilla area. The channel is closed by the Oddi sphincter. The liver daily produces about 800 to 1.000 ml. of bile, of which the main component is bile acids that are synthesized in the liver from cholesterol. The liver plays the main role in the metabolism of sugars, carbohydrates, proteins and fats. Hepatocytes are equipped with a whole range of enzymes which serve for this task. The liver traps fats in the form of lipoproteins in the blood and utilises the rest in various metabolic ways, e.g. for the synthesis of cholesterol which serves as a precursor of steroid hormones. The liver is also able to store fat as energy reserves. In the course of the metabolisation of protein in the liver, amino acids are broken down and the ammonia generated is detoxified by its conversion into urea, which is excreted by the kidneys. The liver provide synthetic majority of plasma proteins, including the components of coagulation and fibrinolytic cascades. The steroid hormones, thyroid hormones and pancreas by-products are all (decomposed) by the liver.[1]

Liver disease leading to failures in the synthesis of lipoproteins can cause lipid metabolism disorders. The fats cannot circulate between the liver and the adipose tissue. The subsequent accumulation of fat in the hepatocytes is reversible, but can lead to their damage. Other manifestations of liver diseases are the slow biodegradation of substances from the external environment, like medications and various chemicals for instance. This disability resulting from liver damage is called the Hepatorenal Syndrome, which usually occurs in the advanced stages of liver failure. Severe liver damage leads to a condition which is called Liver Failure; one of the consequences of hepatic function failure is Hepatic Encephalopathy. (Brain-damage). Mild forms of hepatic encephalopathy manifest themselves in the forms of lethargy and stupor, in more serious cases, as coma.[2,3] Viral infections or toxic substances cause liver damage, to which the organism reacts with an inflammatory response that can be acute or chronic. Acute hepatitis is usually of viral origin. A characteristic of this is diffuse damage by seats of necrosis and the regenerating liver parenchyma. The liver is swollen and may be colored by the bile that the damaged cells cannot secrete enough into the canaliculi, and which may be retained by the swelling of the tissues surrounding the canaliculi. The majority of viral hepatitis is caused by viruses that specifically attack the liver parenchyma. Hepatitis viral infections are also caused a whole range of sources, for example by, the EB virus, herpes viruses, adenoviruses, enteroviruses, or cytomegalovirus. The portal vein carries blood from the digestive tract organs, pancreas and spleen to the liver. The cause of the rise in blood pressure in the portal vein is a breakdown in the hepatic blood flow caused, in most cases, by damage to the liver parenchyma in the course of liver cirrhosis. In the development of portal hypertension, two factors come into play, namely: compression of the capillaries and the increased flux on the hepatic artery. Cirrhosis reduces the liver´s functional capacities not only by parenchymal destruction, but due to the fact that - in portal hypertension situations, blood from the digestive tract leaves the hepatic circulation and reaches the inferior vena cava by alternative routes. Another issue is the so-called Portocaval Anastomoses, which are the vascular connections between the portal blood vessels and upper or lower vena cava. Another symptom of portal hypertension is

ascites, where this has to do with the accumulation of fluid in the peritoneal cavity, while at the same time splenomegaly caused by the increased hydrostatic pressure in the portal circulation system. Complications caused by ascites are pressure on the abdominal organs, difficulty in breathing and rarely, spontaneous bacterial peritonitis. In laboratory tests of blood and urine, the indicators of damage to the liver parenchyma are abnormally decreased or the level of hepatic metabolites is increased. Pathogenic microorganisms damage their host, which can be characterised by some the following mechanisms, such as:

- The adhesion and invasion of cells
- The production of toxins
- The induction of immunopathological reactions

The micro-flora of the liver organs of biological subjects

We differentiate between (autochthonic) indigenous micro-flora, and the body's own micro-flora and (allochtonic) non-indigenous, including microorganisms occurring only temporarily in the intestine. The majority of the bacteria is contained in the colon; and represents a form of "bioreactor" which assists in the breakdown of digestible and indigestible polysaccharides and in the synthesis of micronutrients, like vitamins and short-chained fatty acids. The majority of the bacteria contained in the colon represent a form of "bioreactor" which assists in the breakdown of indigestible polysaccharides and in the synthesis of micronutrients, like vitamins and short-chained fatty acids. The following are considered to be the main causes of intestinal bacterial overgrowth in cirrhotic patients: achylie, decreased secretion of IgA, and malnutrition caused by hepatic dysfunction, or alcoholism. Impairment to the immune mechanisms of the small intestinal mucosa facilitates bacterial overgrowth may be one explanation of recurrent spontaneous bacterial peritonitis in biological objects with liver cirrhosis.

On the contrary, the suppression or eradication of intestinal facultative anaerobic gram-negative bacteria prevents bacterial translocation and SBP not only in cirrhotic laboratory materials in laboratory experiments as well as in patients with cirrhosis of the liver. Also, the slowed-down intestinal motility associated with cirrhotic liver disease facilitates bacterial overgrowth in the gastrointestinal tract of humans.

The main mechanisms leading to bacterial translocation are deficits in local mucosal immune responses, decreased reticuloendothelial phagocytic activity and qualitative neutrophil dysfunction, increased permeability of the intestinal barrier and intestinal bacterial overgrowth.

Verification of cirrhotic laboratory materials in the experiment

Preference was given to the regression function to statistically test value significance:

$$F = \frac{S_R / p}{S_E / (n - p - 1)} \tag{1}$$

Last, but not least, a check is made of the residual values. The corresponding *p*-value indicates that the data is normally distributed. The Null Hypothesis for this test is that the data is normally distributed. The *p*-value indicates that, for a significance level of 0.05, we cannot reject the Null Hypothesis.

Table 1. Regression parameter estimate

General Growth Model: Y=a*Exp(-b*X)+c
Dependent variable: Y Independent variable : 1
Loss Function: Weighted Least Squares
Coefficient of determination : ,99937853 R = ,99968921

	parameter estimates	standard errors	t-value sv = 75	p-value	95% interval	95% interval
a	18,65420	0,054450	342,5961	0,00	18,54574	18,76267
b	0,00993	0,000076	130,0065	0,00	0,00978	0,01008
c	19,55823	0,036692	533,0311	0,00	19,48514	19,63133

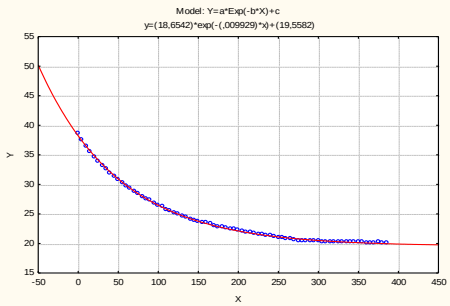

Fig.1 Chart of the data interpolating the curve

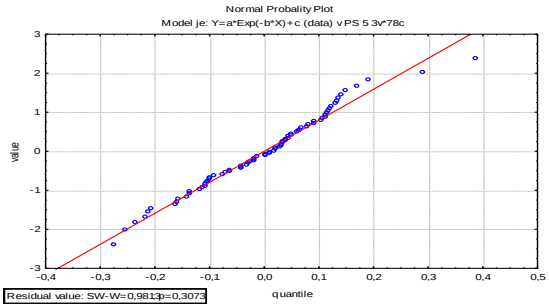

Fig. 2 Chart of residue dependence on the predicted values

Summary

This paper presents the primary principles of functional regression methods with bacterial translocation to determine the time of death of biological material. An important aspect is the issue of the exact determination of measurement uncertainties in the process of bacterial translocation measured in the liver parenchyma (i.e. lobus sinister and lobus dexter). Analysis of regression parameters and normality residue of the data obtained led to the drawing of conclusions which lead to the formulation of concrete processes used to determine the time of death of biological material by means of the polynomial regression method, and which instigates the drafting of strategic measures for its practical application(s). The research is conducted in the Industrial Property Ownership - IPO Register, under No. PV 2012-124.

References

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