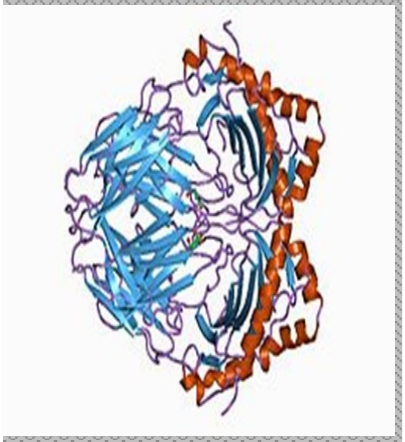

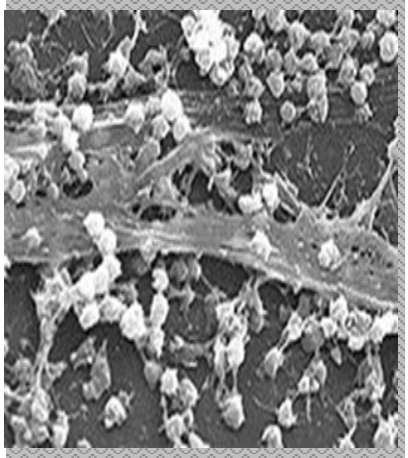

EMGEN Newsletter

Vol. 5, Issue 4

IN THIS ISSUE:

1. **Article, P 2**
2. **Training, P 8**
3. **Trends, P 13**
4. **News, P 17**
5. **Book Alert, P 19**
6. **Announcement, P 20**
7. **Cover pictures description, P 22**

Eastern Mediterranean Health Genomics and Biotechnology Network (EMGEN) was created in 2004 with collaboration of representatives of selected centers of excellence in (health related) molecular biology, biotechnology & genomics in the Eastern Mediterranean region by recommendations and efforts of WHO/ EMRO. Sponsored by Iran Biotechnology Development Council.

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Article

The article below, entitled "Estrogen Receptor-α A908G (K303R) Mutation and Breast Cancer Risk" was published in "International Journal of Clinical and Experimental Medicine", Vol. 6(1), pp. 39-49. The study was carried out by Dr. Sakineh Abbasi working at the Department of Medical Biotechnology, School of Medicine, Tehran University of Medical Sciences, Tehran, Islamic Republic of Iran.

Dr. Sakineh Abbasi

Introduction

Genetic mutations in premalignant breast trauma can affect malignancy advancement or the behavior of other diseases. The primary risk factors for breast cancer are abnormality in hormones or reproductive factors which enhance disposal to estrogen. A point mutation in estrogen receptor-α (ER-α) as A908G (Lys303→Arg) was at first involved to estrogen breast hyperplasia. At the moment, the researches reached little data regarding ER -α gene expression, mutational abundance, and allelic types in breast cancer across Asia-Caucasians (Iranian), particularly those who inhabit their domestic homeland. Hence, this study was designed to screening a sequel of recently recognized aggressive breast cancer from patients referred to the Imam Khomeini Hospital Complex, Tehran; a population-based case study of breast cancer in Iranian people for ER-α A908G point mutations by applying a composition of Single-Strand Conformational Polymorphism (SSCP) analysis with help of 33P-cycle DNA sequencing method.

Methods

A 329 bp fragment of 336 bp of exon 4 was amplified with reverse (5'-GCTGCGCTTCGCATTCTTAC-3') and forward (5'-ACCTGTG TTTTCAGGGATACGA-3') primers, based on the existing protocols. Intended to recognize the mutation at codon 303 of ER-α amid Iranian society, the plan was to sieve samples by use of the SSCP-PCR method. PCR samples represent different band shifting patterns by SSCP. These PCR samples were separated on agarose gel using a DNA Extraction Kit. Sequencing was carried out on both the forward and reverse DNA strands by use up 33P- cycle sequencing method. All the mutations were confirmed (and the feasibility of mutation artefacts was omitted) by sequencing of a second, singly amplified PCR product. In addition, at least 5% of the SSCP-negative samples were sequenced (n = 46), but no mutation was observed.

Results

By numbers, considerable abundance were attained solely for risk factors, age at menstruation 12 years old and below, those who reside in the southern region of Iran; in addition, types of blood groups including A, B and O blood groups, in ABO blood types (Table 1).

Across all of these risk factors just people with family history of breast cancer and lymph node metastases showed statistically significant distinction ($P<0.05$) among various genotypes (Table 2). The heterozygote genotype (AAG/AGG) observed only in patients with cancer (10.7%). Accordingly, the allele 1 (AGG) in codon 303 was existed merely in patients with cancer (5.3%). The allelic redundancy of allele 1 (AGG) in codon 303 was notably premiere (fourteen-fold) in the cancer patients with a cancerous relative (28.9%) than those without a family member with breast cancer (1.9%) (Table 3). The guesstimated risk of cancer in normal genotype patients without a cancerous relative (94.0%) is higher than patients with family history of breast cancer (6.0%); but, the calculated risk was lower (more than two-fold) for heterozygote persons in the mutation of codon 303, without a cancerous relative (31.2%) than peoples with family history of breast cancer.

Discussion

The results of this study acknowledged the presense of the ER-α gene A908G mutation in Iranian women progressive breast cancer. By the way, the redundancy of A908G or K303R mutation was higher (10.7%) compared with the other research results, which suggests that mutations of ER-α gene K303R happen at less redundancy rate in breast tumor (around 6%).

In the first assessment of independence of the ER-α gene A908G mutation with demographic and clinical specifications of breast cancer instances among Iranian people, including age at menstruation below 12 years old in comparison with age at menstruation above 12 years old, from ABO blood types, type B and O and among all eight various strains, peoples of Fars, were considered just in heterozygote cancer patients with a statistically significant redundancy in heterozygote cancer patients.

The primary risk factors for breast cancer are abnormality in hormones or reproductive factors which enhance disposal to estrogen. The significance of estrogen in breast cancer formation is further supported by studies representing the incidence of marked variations in estrogen signaling and expression of the two estrogen receptors (ERs) including ER-α and ER-β within breast tumor emersion and advancement. Data acquired from other epidemiologic studies of breast cancer propose that tumor subsets arranged based on certain somatic or protein expression variations may be affiliated with particular etiologic risk factors.

The evaluated risk factors for normal genotypes without family history of breast cancer (94.0%) was very higher than people with family history of breast cancer (6.0%); but, the evaluated risk for heterozygote patients in codon 303 mutation, without any case of cancer in relatives (31.2%) was much lower than patients with family history of breast cancer (68.8%), compared with controls. Breast cancer cases in Iranian people with ER-α A908G mutation-positive tumors were more likely to have a first-degree family of breast cancer; while, the mutation-negative cases were not; this discovery was based on the case comparisons.

Eventually, in total, it was noted that: 1) The presence of the ER-α A908G point mutation in aggressive breast tumors may have significant concept for etiology, anticipation and direct dependency for increasing risk of advancing breast cancer, even in heterozygote genotype; 2) The redundancy of this point mutation of breast cancer in people with the family history of breast cancer is more possible (A→G) than those without family history of breast cancer, and finally; 3) The greater the abundance of mutant allele, the lower the possibility of LN metastasis in Iranian society. Minor but statistically remarkable correlations were found among allelic repartition and familial revelation of breast cancer.

Conclusions

This findings indicate that ER-α codon 303 mutation is associated with different sights of breast cancer in Iran. ER-α genotype might demonstrate a suitable marker for predicting breast cancer developing later in life.

Table 1. Genotypic repartition redundancy of codon 303 in exon 4 mutation of ER-α gene and selected demographic specifications and primary risk factors in the target population..

Characteristic	Group	Normal [a]		Heterozygote [b]		Test result
		Frequency	Percent	Frequency	Percent	
ABO blood groups						
A	Case	24	88.9	3	11.1	c^2=5.932 *P*=0.015
	control	43	100	-	-	
	total	67	95.7	3	4.3	
B	Case	10	71.4	4	28.6	c^2=10.957 *P*=0.001
	control	35	100	-	-	
	total	45	91.8	4	8.2	
AB	Case	6	100	-	-	-
	control	16	100	-	-	
	total	22	100	-	-	
O	Case	94	91.3	9	8.7	c^2=7.753 *P*=0.005
	control	53	100	-	-	
	total	147	94.2	9	5.8	
Total	Case	134	89.3	16	10.7	
	control	147	100	-	-	
	total	281	94.6	16	5.4	
Race						
Arab-Armani	Case	3	100	-	-	-
	control	-	-	-	-	
	total	3	100	-	-	
Fars	Case	50	83.3	10	16.7	c^2=19.134 *P*=0.001
	control	88	100	-	-	
	total	138	93.2	10	6.8	
Lor – Kurdish	Case	16	88.9	2	11.1	c^2=1.701 *P*=0.192
	control	9	100	-	-	
	total	25	92.6	2	7.4	
Turkish	Case	44	95.7	2	4.3	c^2=2.497 *P*=0.114
	control	39	100	-	-	
	total	83	97.6	2	2.4	
Gilaki-Mazani	Case	21	91.3	2	8.7	c^2=1.623 *P*=0.203
	control	11	100	-	-	
	total	32	94.1	2	5.9	
Total	Case	134	89.3	16	10.7	
	control	147	100	-	-	
	total	281	94.6	16	5.4	

[a] Genotype normal, AAG/AAG, [b] Genotype heterozygote, AAG/AGG.

Table 2. Genotypic redundancy of codon 303 in exon 4 mutation of ER-α gene and selected demographic properties and primary risk factors in the breast cancer group.

Characteristic	Normal		Heterozygote		
	Frequency	Percent	Frequency	Percent	Test result
Onset age of breast cancer (years)					
<40	43	89.6	5	10.4	c^2=0.005
>/=40	91	89.2	11	10.8	*P*=0.946
Total	134	89.3	16	10.7	
Family history of breast cancer					
First-degree family affected	8	42.1	11	57.9	c^2=33.518
Not affected	126	96.2	5	3.8	*P*=0.001
Total	134	89.3	16	10.7	
ER expression in breast cancer tissue					
Positive	34	85.0	6	15.0	c^2=4.6
Negative	86	93.5	6	6.5	*P*=0.1
Not studied	14	77.8	4	22.2	
Total	134	89.3	16	10.7	

Table 3. Allelic redundancy of ER-α exon 4 mutation at codon 303 (AAG→AGG) in the target population.

Characteristic		ER-α Alleles	
		0	1
Breast cancer			
Case	(n=150)	284(94.7%)	16(5.3%)
Control	(n=147)	294(100%)	-
		c^2=16.114 , *P*=0.001	
Age at menarche at (years)			
</=12	(n=60)	111(92.5%)	9(7.5%)
>12	(n=90)	173(96.1%)	7(3.9%)
		c^2=1.86 , *P*=0.173	
Onset age of breast cancer			
</=40	(n=48)	91(94.8%)	5(5.2%)
>40	(n=66)	126(95.5%)	6(4.5%)
After menopause	(n=36)	67(93.1%)	5(6.9%)
		c^2=0.535 , *P*=0.765	
ABO blood groups			
A	(n=27)	51(94.4%)	3(5.6%)
B	(n=14)	24(85.7%)	4(14.3%)
AB	(n=6)	12(100%)	-
O	(n=103)	197(95.6%)	9(4.4%)
		c^2=4.838 , *P*=0.184	

Table 4. Calculated risk for selected demographic properties and primary risk factors with ER-α exon 4 mutation at codon 303 in various genotypes.

Genotype	**Yes n=150**	**No n=147**	***P* value**	**OR (95% CI)**
Normal [a]	134(47.7%)	147(52.3%)	0.001	1.0(reference)
Heterozygote [b]	16(100%)	-		-
First- degree family history of breast cancer	**Affected n=19**	**Not affected n=131**	***P* value**	**OR (95% CI)**
Genotype				
Normal	8(6.0%)	126(94.0%)	0.001	1.0(reference)
Heterozygote	11(68.8%)	5(31.3%)		0.029(0.008-0.103)
Lymph node metastases	**Yes n=23**	**No n=127**	***P* value**	**OR (95% CI)**
Genotype				
Normal	23(17.2%)	111(82.8%)	0.017	1.0(reference)
Heterozygote	-	16(100%)		-

[a] Genotype normal , AAG/AAG, [b] Genotype heterozygote , AAG/AGG.

BIOMARKERS

Multiple sclerosis

Multiple Sclerosis (MS) is an advanced autoimmune derangement of the central nervous system (CNS), that approximately over 2.5 million people worldwide suffer from it. MS has been extensively investigated, however many challenges still remain in regards to treatment, diagnosis, and prediction, which is classified into three principal types of clinical courses:

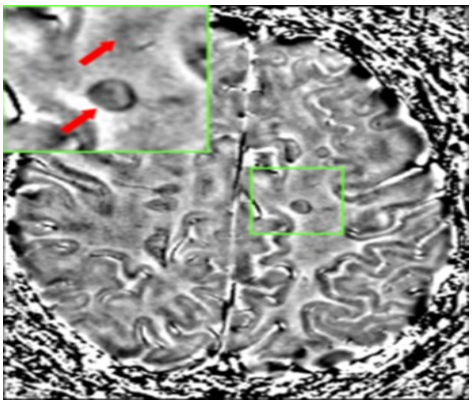
1. Relapsing–remitting (RRMS) 2. Primary progressive (PPMS) 3. Secondary progressive (SPMS).

Although the etiology of the disease is unknown, evidence suggests that the disease may result from a complex interaction between the environmental factors, the genetic context that specify individual aptitude, and the immunological and physiological settings of the individual. Genetics and environmental stimulus have a major role in the development of MS. Epstein Barr Virus infection, childhood obesity, smoking, and low vitamin D levels, including the environmental factors are associated with the disease.

MS Biomarkers

Biomarkers are indicators that measure and evaluate biological and pathogenic processes, or pharmacological reaction to a remedial intervention. An authentic and stringent biomarker should be able to link to treatment, control, and diagnosis of MS through inflammatory activity, and the degree of neuronal decadence and demyelination/remyelination, in order to have a precise picture of the disease situation.

There are three main regions of research into biomarkers for MS: identification of disease specific biomarkers that forecast which people are at risk of developing MS; determining advanced biomarkers that can forecast individuals that are at high risk of developing intense attacks or advanced disease; and, finding volunteer people who may respond to specific treatments in order to perform an individual care program for demonstrating the maximum effectiveness and minimum side effect.



Picture: A Gradient-echo phase MRI scan of brain showing an iron deposit in a white matter lesion in an MS patient.

Clinical use of Biomarkers

There are three biomarkers for management of diagnosis and treatment of MS:

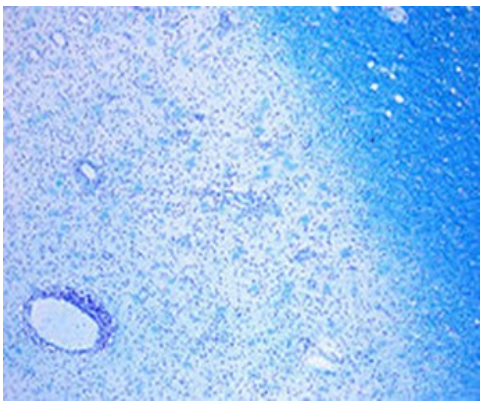
1. Oligoclonal bands and IgG ratio.
2. MRI and MRI contrast enhancing lesions.
3. JC viral antibody titers.

Diagnostic biomarkers

Diagnostic biomarkers can diagnose patients who have MS from patients with other neurological derangements, or from healthy people. Indeed, white-matter lesions in typical MS can be seen in many other neuroinflammatory conditions, such as neurosarcoidosis, neuroborreliosis, Sjogren's syndrome, and systemic lupus erythematosus. Diagnosis of oligoclonal IgG bands in the CSF in clinical practice is the only reliable biomarker for detection of MS. Therefore, MRI of brain and spine plus OCGB arrangement in CSF shows the inflammatory and demyelinating nature of the MS and is an important tool in diagnosis of this disease. Also, serum antibodies against particular antigens in several neuro-immunological diseases such as myasthenia and para neoplastic derangements have been manufactured. The importance of autoantibodies as diagnostic biomarkers have been emphasized following the finding of a serum pathogenic antibody targeting the major water channel of astrocyte aquaporin-4, distinguishing neuromyelitis optical (NMO), also known as Devic's disease, from MS.

Genetics as biomarkers

Genome wide association scans have now defined over 100 genetic diversities that are related to MS with that number likely to rise to over 200. GWAS (genome-wide association study) are case control studies designed to present diversities that confer a modest risk of genetic illness. These studies compare the frequency of genetic diversities in a healthy control society with the frequency seen in MS patients. The common genetic diversities related to MS and other autoimmune diseases commonly have a small contribution to disease. Genomic screens have a number of benefits that make them ideal for biomarker studies. First, they can be performed fast and cheap from a small amount of blood. This negates the problems of requiring lumbar puncture for acquiring CSF and makes standardization among sites simple. Secondly, GWAS provide a detailed, unbiased view for novel biomarkers. Swiftly emerging genomic technologies are making genetic screens cheaper and more ordinary for many illnesses.



Picture: Demyelination in MS. Discoloration in the region of the lesion can be seen (Original scale 1:100).

Potential biomarkers for use in MS

1. Markers of neurodegeneration, CNS neurofilaments and GFAP

CNS neurofilaments (Nfl), involve neuro filament weighty, medium, and light chains that both the weighty chain and light chain are related to axonal damage in MS and α-internexin are released after axonal damage. In the present study the focus was on the light chain (NFL) that being a more authentic, consistent and reliable marker in MS. In addition to this, various other markers of neuronal and glial cell injury have been illustrated to be elevated in MS patients compared to healthy individual. S100B, tau, NCAM, NGF, CNTF and ferritin expression in the CSF also have been suggested as potential biomarkers related to MS.

2. sCD163

CD163 is a monocyte/macrophage special membrane marker.

3. YKL-40 (Chitinase-3-like 1)

YKL-40 (Chitinase-3-like 1) is a glial activation marker that is also represented on activated macrophages, vascular smooth muscle cells, airway epithelia, and chondrocytes.

4. CXCL13

CXCL13 is a B cell chemo attractant that interacts with CXCR5 on B cells and is needed for the expansion of B cell follicles and secondary lymphoid structures.

5. Anti-microbial antibodies

Antibodies against varicella zoster (MRZR), rubella, and measles are existent in the CSF of ~80% of MS patients that measles antibody viral titers in MS patients were first defined in 1973.

6. miRNA and mRNA

mRNA and miRNA can be separated from peripheral blood mononuclear cells, purified immune cell subsets, serum, and CSF. miRNAs have a significant role in numerous phenomena including growth, organogenesis, and homeostasis.

7. Myelin-reactive T cells

Both CD4 and CD8 T cells are existing in MS lesions and are believed to have a fundamental role in disease progression.

8. KIR4.1 antibodies

Neuromyelitis optical (NMO), a derangements characterized by optic neuritis and transverse myelitis, was originally thought to be a subset of MS.

9. Serum osteopontin

Osteopontin (OPN) is an early activation marker on T cells with a role in T cell stimulation and IFNγ explanation. OPN is highly expressed within MS lesions and is significantly higher in MS patient`s blood and CSF than normal controls.

10. Micro biome-associated lip peptides

The micro biome in humans and mice has been demonstrated to have substantial impact on disease.

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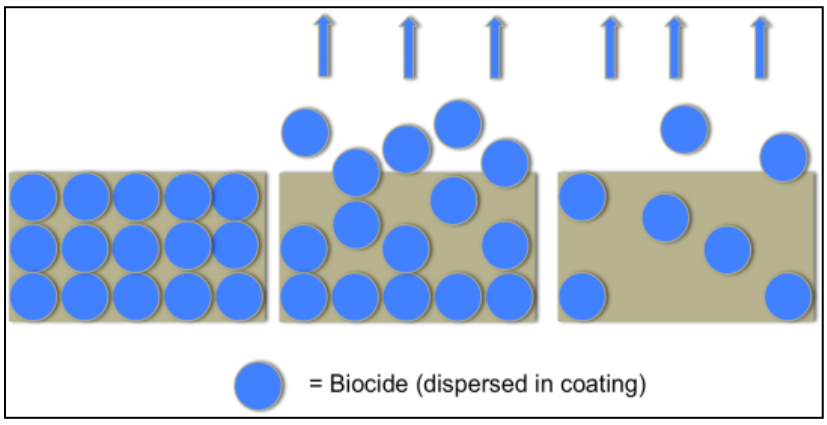
ANTIBACTERIAL SURFACES

Despite of considerable growth in the nanobiotechnology and nanofabrication techniques, the effort to design and construction of new antibacterial surfaces and development of existing antibacterial surfaces as an integral component of advanced biomaterials remains a significant research preference. The colonization of surfaces by bacteria and expansion on surfaces was diagnosed in the 1930. It was found that bacteria prefer to dwell on solid surfaces instead of dwelling on planktonic substrate. Microorganisms, which millions of years are living on our planet, inhabiting surfaces and cause destructive effect are changing the function of specific interfaces, such as those found in petroleum pipelines and aquatic flow systems, contact lenses, textiles, and medical implants. Recently, the formation of biofilms has been widely investigated in an attempt to develop several surface modification methods to inhibit or reduce the amount of bacterial adhesion using antibiotics, biocides, and surface treatment processes. In order to eliminate or significantly decrease the amount of bacterial attachment and prevention of biofilm formation on these surfaces, or on the improvement of the efficiency of available antibacterial surfaces, several attempts have been focused on the building of new surfaces, for example, the application of surface coatings, or modification and alteration of the surface architecture. In this article, it is suggested that antibacterial surfaces with attention to the effect that these surfaces have on biological systems, should be classified as being either antibiofouling or bactericidal. A group of antibacterial surfaces according to the surface chemistry modifications or surface coating to which they have been subjected can be categorized; for example, surface functionalization, polymerization, and modification to the surface physical modification. Much endeavors to design a new classification of antibacterial surfaces.

Picture: Biocide release over time.



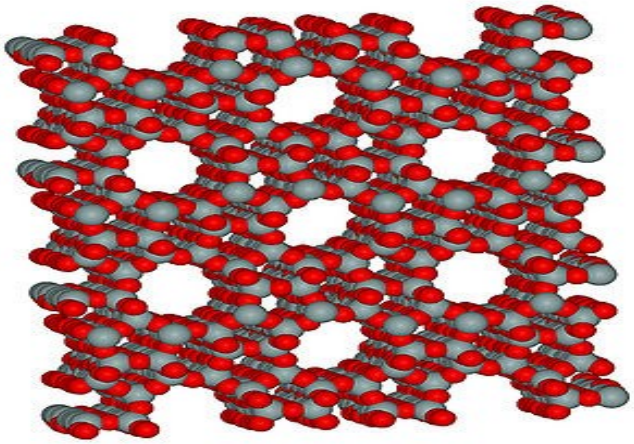
Artificial antibacterial surfaces

A number of advanced surface modification techniques and traditional methods have been extensively utilized in formation of artificial antibacterial surfaces. These surfaces contain a series of polymer- and nanoparticle-based surfaces. A number of these artificial surfaces have displayed a bactericidal or anti biofouling effect. Silver-based bactericidal surfaces mainly contain silver-doped, coated, silver-containing polymers, silver nanoparticles, or silver thin films.

Antimicrobial surface

An antimicrobial surface possess an antimicrobial factor that prevents or reduces the ability of microorganisms to grow on the surface of an organic material such as a corpus. Such surfaces have been widely investigated for possible use in varied applications including clinics, industry, and others. Most important application of antimicrobial coatings have been reported in the sanitation and health perimeter for sterilization of medical devices to inhibit hospital associated infections. In addition, in medical devices, linens and clothing a suitable environment exists for bacteria, fungi, and viruses to grow; also, when it contacts with the human body allows the infectious pathogen to transmit. An innovation in antimicrobial surfaces is the finding that copper and its alloys (bronzes, brasses, cupronickel, and others) are natural antimicrobial materials that have inherent abilities to destroy an extensive series of microbes.

Metals such as silver (Ag), gold (Au), copper (Cu) and zinc (Zn) having been known for antimicrobial coating and activity. Silver has been used to inhibit microbial colonization of prostheses, catheters, and human skin. Silver-based coatings are widely used in medical implants due to the silver ions discharged from the surface are bactericidal against both Gram-positive and Gram-negative bacteria. Copper alloys, operated in doorknobs and other surfaces, displayed an *in vitro* antimicrobial effect against *Escherichia coli* O157, *Clostridium difficile* and methicillin-resistant *Staphylococcus aureus* (MRSA), while equivalent stainless steel surfaces did not. Copper and zinc amalgams have proven useful in dental materials.



Picture: Structure of zeolite. The spongy composition is suitable for accommodate ions such as Ag+.

Antimicrobial features of copper

Antimicrobial features of copper and its alloys (brasses, bronzes, and others) became understood in the nineteenth century. In addition, in several copper medicinal researches, it was also demonstrated that water contains copper or transported in copper transmission systems was of better quality than water contained or transported in other materials. Researchers are actively demonstrating the innate abilities of copper alloy to destroy a wide series of microbes. Antimicrobial copper-alloy touch surfaces prevent often the spread of disease-causing microorganisms. This is especially true in sanitation and health facilities, where harmful bacteria, viruses, and fungi colonize and persist on doorknobs, push plates, railings, HVAC systems, and other equipment. These microorganisms can frequently live on surfaces for long time. They have intrinsic properties to kill a wide series of harmful microbes rapidly – often in two hours or less – and with a high grade of efficiency. That touch surfaces are made with copper alloys, the reduced transmission of disease-causing microbes can decrease patient infections in hospitals by as much as 58%.

Picture: Native copper (~4 cm in size)

Efficacy of copper surfaces

Researches represented that copper alloy surfaces kill over 99.9 % *E. coli* O157 after just 1–2 hours. On stainless steel surfaces, the microbes can live for weeks.

E. coli O157 on the copper alloys is faster at room temperature than at chill temperature. The bacterial kill rate of copper alloys increased with increasing copper content of the alloy. This is further indication of copper's inherent antibacterial abilities.

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NEW DRUG DELIVERY APPROACH HOLDS POTENTIAL FOR TREATING OBESITY

Researchers at MIT and Brigham have created nanoparticles that can deliver anti-obesity drugs instantly to fat tissue. Obese mice treated with these nanoparticles lost 10 percent of their weight during 25 days, without showing any harmful side effects.

The medication works by changing white adipose tissue, which is built of fat-hoarding cells, into brown adipose tissue, which burns fat. The medication also agitates the growth of new blood vessels in fat tissue, which positively reinforces the nanoparticle targeting and helps in the white-to-brown changing.

Targeting fat

Langer and his teammates have previously shown that boosting the growth of new blood vessels, a procedure recognized as angiogenesis, can assist transform adipose tissue and terminate to weight loss in mice. Anyway, drugs that boost angiogenesis can be injurious to the rest of the body. To solve that, researchers tried to use the nanoparticle drug-delivery approach they have expanded in recent years to cure cancer and other diseases. By targeting these particles to the disease site, they can deliver a strong dose while decreasing the drug`s reposition in other areas.

The researchers made the particles to transport the drugs in their hydrophobic cores, bound to a polymer recognized as PLGA, which is applied in a lot of other drug delivery particles and medical devices. They packaged two dissimilar drugs inside the particles: rosiglitazone, which has been accepted to treat diabetes and an analog of prostaglandin. Both drugs activate a cellular receptor identified as PPAR, which agitates angiogenesis and adipose changing.

The outer membrane of the nanoparticles comprises of one more polymer, PEG, embedded with targeting molecules that direct the particles to the right destination. These targeting molecules attach to proteins found in the lining of the blood vessels that enclose adipose tissue.

The researchers used the particles in mice that had become fat after being fed a high-fat diet. The mice lost about 10 percent of their weight, and their rate of cholesterol and triglycerides also decreased. The mice also became more sensitive to insulin. Obesity often causes insulin impassibility, which is a risk factor for type 2 diabetes.

Reference: https://www.sciencedaily.com/releases/2016/05/160502161116.htm

EXPERIMENTAL DRUG AGAINST HEPATITIS C SLOWS DOWN ZIKA VIRUS INFECTION IN MICE

Virologists from KU Leuven, Belgium, have revealed that an experimental antiviral drug against hepatitis C decreases the growth of Zika virus in mice. The research group was conducted by Professor Johan Neyts from the Laboratory of Virology.

"The virus can spread by the forest mosquito. Approximately 20 percent of the communities who are infected usually get ill," stated Professor Neyts. "The most familiar indications, which last about a week, are joint and muscle pain, rash, red eyes, fatigue and fever. Some of infected people continue to develop Guillain-Barré Syndrome, which induce muscle infirmity and interim palsy. In some cases, the patient requires to be put on a ventilator."

"One of the important concerns is that, pregnant women with the disease can transfer the virus to the embryo," Neyts mentions. "As a consequence, some babies will born with microcephaly, a disorder of the central nervous system (CNS) whereby the child`s skull and brain are too small. In serious cases, these children grow up with critical mental and physical inabilities." Following immense prevalence of the virus on islands in the Pacific, the virus developed rapidly in Central and South America in 2015 and 2016. In beginning of this year, the World Health Organization (WHO) announced the state of emergency to include the epidemic as quickly as possible. Finally, there is no vaccine or antiviral medicine accessible to prevent or treat an infection at present.

Zika virus has a close relationship to the hepatitis C virus. The research team evaluated the effect of some inhibitors of the hepatitis C virus on the growth of the Zika virus in human cells. They mentioned they found at least one experimental medicine that is useful for treating the Zika virus infections. Following this, the researchers investigated the inhibitory effect of anti hepatitis C virus drugs on Zika virus in mice with a defect in their innate immune system. The infected mice with Zika virus demonstrate some symptoms which are similar to human patients. Treating the infected mice with the hepatitis C virus inhibitor causes an obvious lag in virus -caused symptoms.

Reference: https://www.sciencedaily.com/releases/2016/05/160517094212.htm

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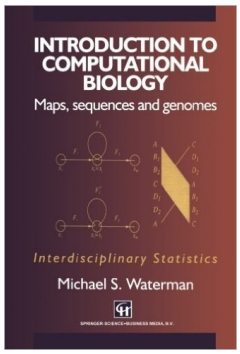

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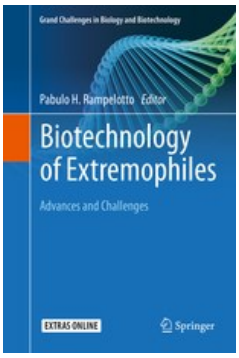

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TITLE: Myelin basic protein

Myelin basic protein (MBP) is a second most profuse protein in central nervous system myelin. The myelin sheath is in the form of a multi-layered membrane. MBP is an important component of the myelin sheath which MBP keeps the correct composition of myelin, work together with the lipids in the myelin membrane. MBP is the widely studied myelin protein in MS that is essential for the normal activity of the nervous system. MBP accounts for about one third of total CNS (Central Nervous System) myelin protein, and CNS MBP concentrations rise in reply to neuronal damage.

MBP was primarily arranged in 1971 after separation from myelin membranes. Since that time, knockout mouse shortage in MBP that showed decreased amounts of CNS myelination and a progressive disorder diagnosed by tremors, seizures, and premature death have been increased. The gene for MBP is on chromosome 18; the protein centralize to the CNS and to different cells of the hematopoietic system.

The pool of MBP in the central nervous system is very various; with several splice variants being represented and a great number of post-translational alterations on the protein, that consist of phosphorylation, methylation, deamination, and citrullination. These forms vary by the presence or the nonexistence of short peptides in different internal sites in the series. Overall, the main form of MBP is a protein of about 18.5 KD (170 residues).

Reference: https://en.wikipedia.org/wiki/Myelin_basic_protein

TITLE: Biofilm

A group of microorganisms attach to a surface and form biofilms. A biofilm consists of many bacteria. Often these sticky cells are embedded in a matrix of extracellular polymeric substances (EPS). Biofilm extracellular polymeric substance, which is referred to as slime, is a polymeric mass commonly composed of extracellular DNA, proteins, and polysaccharides.

Biofilms may form on a wide variety of surfaces, including living tissues, hospital and industrial settings or natural systems. The microbial cells are formed in a biofilm are physiologically separate from planktonic cells of the same organism, which in return, are single-cells that may float in a liquid medium.

Bacteria constitute a biofilm in reply to several factors, which may comprise cellular diagnosis of specific or non-specific attachment places on a surface, nutritional signals, or in some suitcases, by disposal of planktonic cells to sub-inhibitory concentrations of antibiotics.

When a cell changes to the biofilm mode of growth, it undergoes a phenotypic modification in performance in which great sets of genes are differentially regulated.

Reference: https://en.wikipedia.org/wiki/Biofilm

TITLE: Drug delivery

Drug delivery is the technique of management pharmaceutical compound in the body for obtain a positive effect in treatment. Drug delivery is important and useful for the treatment of many diseases. This technologies shift drug release profile, absorption, circulation and exclusion for the advantage of improving product efficacy and safety, as well as patient easement and compatibility.

Drug release is from: diffusion, degradation, swelling, and affinity-based mechanisms. Most common paths of management include the preferred non-invasive per oral, inhalation paths, trans-mucosal (nasal) and topical (skin). They are easy to formulate into diverse devices for transmission a variety of drug classes, such as peptide and protein, antibody, vaccine and micro molecules. Many protein and peptide drugs have to be delivered by injection or a nanoneedle groups. Many vaccines are based on the delivery of protein drugs and are often done by injection.

Current attempts in the region of drug delivery include the growth of targeted delivery in which the drug is only active in the target region of the body (for example, in cancerous tissues). In order to achieve efficient targeted delivery, the intended system must evade the host's defense mechanisms and circulate to its intended site of action.

Reference: https://en.wikipedia.org/wiki/Drug_delivery