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Deutsche Veterinärmedizinische Gesellschaft e.V. (DVG)

Arbeitsgebiet Lebensmittelhygiene

Vorsitzender: Univ.-Prof. Dr. Dr. h.c. Andreas Stolle

DVG - AG Lebensmittelhyg., Schönleutnerstr. 8, 85764 Oberschleißheim

Certificate

This is to certify that Dr. UGUR GOGUS held an oral presentation on the topic "SHELLFISH POISONING MATTERS" on September 27th 2007 during the conference "48. Arbeitstagung des Arbeitsgebietes Lebensmittelhygiene der Deutschen Veterinärmedizinischen Gesellschaft".

Prof. Dr.Dr. h.c. A. Stolle

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Saal „Olympia" - Fischhygiene

Gogus, U. (Ankara/TR) **11.10 Uhr**
Shellfish poisoning matters

Brenner, M., S. Ramdohr, M. Stede, S. Effkemann, **11.25 Uhr**
E. Bartelt, V. Etzel, A. Koehler (Bremerhaven, Cuxhaven)
Projekt *Mytifit* - Untersuchungen zur Zucht- und Verzehrsfähigkeit von Miesmuscheln (*Mytilus edulis*) aus Offshore Windparks

Özden, Ö., N. Erkan, M.C. Deval (Istanbul/TR) **11.40 Uhr**
Jahreszeitlicher Vergleich der Schwermetallakkumulation bei zwei Muschelspezies im Marmara-Meer, Türkei

Diskussion **11.55 Uhr**

Mittagspause **12.10 Uhr**

Foyer 1.OG - Posterausstellung **14.00 Uhr**

Institut für Hygiene und Technologie der Lebensmittel tierischen Ursprungs

Vorstand: Univ.-Prof. Dr. Dr. h. c. Andreas Stolle

LMU Tierärztliche Fakultät

Fachausstellung 2007

48. Arbeitstagung des Arbeitsgebietes Lebensmittelhygiene der DVG zusammen mit der Sektion Lebensmittel tierischer Herkuft in der Österreichischen Gesellschaft für Tierärzte und der Schweizerischen Tierärztlichen Vereinigung für Lebensmittelsicherheit

Dienstag, 25. September bis Freitag, 28. September 2007

in Garmisch-Partenkirchen

Sehr geehrte Damen und Herren,

wir würden uns freuen, Sie im Rahmen der Fachausstellung

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Deutsche Veterinärmedizinische Gesellschaft e.V. (DVG)

Arbeitsgebiet Lebensmittelhygiene

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Herrn
Dr. Ugur Gogus
Vocational school of higher education - food technology program
Ankara

Oberschleißheim, 04.06.2007

48. Arbeitstagung des Arbeitsgebietes Lebensmittelhygiene der DVG vom 25.09. bis 28.09.2007 in Garmisch-Partenkirchen

Sehr geehrter Herr Kollege,

der Vorstand des Arbeitsgebietes Lebensmittelhygiene hat in seiner Sitzung am 14.05.2007 in München den von Ihnen angemeldeten Beitrag

„Shellfish poisoning matters"

als Vortrag am **27.09.2007** um **11.10 Uhr** angenommen. Die vorgesehene Redezeit von 15 Minuten darf **unter keinen Umständen überschritten** werden.
Bei Power-Point-Präsentationen ist folgendes zu beachten: Vers. Office 2000 oder darüber auf CD-ROM/Diskette, eigener Laptop nur in Ausnahmefällen bei rechtzeitiger Rücksprache (mind. 2 Std. vor Präsentation) mit Frau Dr. Thiel oder Frau Dr. Sperner.

Ein Proceeding-Band wird seit letztem Jahr nicht mehr erstellt. Ausgewählte Beiträge werden im „Archiv für Lebensmittelhygiene" veröffentlicht.

Vom Referenten bzw. Referentin werden keine Tagungsgebühren erhoben. Die Tagungsunterlagen bitten wir, im Tagungsbüro in Empfang zu nehmen.
Für die Quartierbestellung bietet die Kurverwaltung GAP die Möglichkeit an, über ihre Homepage (www.garmisch-partenkirchen.de) zu buchen. Telefonische Zimmerreservierung kann unter der Nummer 08821 - 180700 erfolgen.

Für Ihre Unterstützung möchte ich Ihnen als Vorstand des Arbeitsgebietes Lebensmittelhygiene im Voraus herzlich danken. Wir freuen uns, Sie in Garmisch-Partenkirchen begrüßen zu können.

Mit freundlichen Grüßen

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SHELLFISH POISONING MATTERS

Abstract

Water is one of the most common vehicles in food poisoning and infections. Some *Vibrio* species and Shellfish toxins may be expressed as the most important ones, in waterborne diseases. Several microscopic marine algae are notoriously poisonous to human who consumes them by the means of shellfish. These organisms are tiny, single celled plants which look like plants on land, capture and use the sun's energy to grow. The shellfish toxin associated poisonings are due to the consumption of contaminated shellfish with these harmful algae, especially during 'red tide'; discoloration of water as a result of the various pigments in algae blooms. Effect mechanisms differ according to the type of the biotoxin such as *Prorocentrum minimum* toxins, TTX (tetradotoxines) and ST(Saxitoxines) which block sodium channels whereas brevetoxins leads to uncontrolled Na+ influx into the cell. The clinical view, followed by the consumption of contaminated shellfish, is severe in many occasions which are characterized with gastrointestinal, upper respiratory and pulmonary and neurological disorders. In spite of their importance in human and animal health, unfortunately, we still face with a lack of data in 'bacteria and algae' relation to form shellfish poisoning which still remains a paradox. The question still remains to be highlighted whether the toxin producer is the algae itself or the bacteria which use these algae as host. Unlike this paradox, we have got enough detailed data as related to their chemical structures and contamination pathways. Another problem waiting for us is the deficiency of bioassays in determination and lack of standardization of monitoring systems in seaside countries which requires urgent studies and improvements. All bioassays which are presently used seem to have limited sensitivity, high cost, kit depending and interference problems. The deficiencies of marine bioassays in determination and selectivity may result in greater problems especially in the consideration of global warming which raises surface water temperatures while decreases deep temperatures which may stimulate the growth of some harmful surface and deep water algae species. We have neither standardization in shellfish toxin monitoring nor high sensitive biotoxin determination and protection in most countries except Italy, New Zealand and Ireland. Some countries still monitor only one or two algal species. Except Italy, others close their harvest areas only when the toxins have been detected in shellfish. However, all seaside countries should close their harvest areas when the numbers of cells exceed certain concentrations. It is noteworthy that canning, heating, fermentation. ultraviolet and chlor do not destroy shellfish toxins, whereas CO_2 and acetic acid super critical atmosphere is found effective in depuration (Gonzales et al. 2002). High Hydrostatic Pressure as combined with CO_2 and acetic acid super critical atmosphere, HHP and acidity combined and HHP alone applications may be performed in very near future to highlight whether the concerned applications have got any destructive effect on marine origin biotoxines. Education of people in the first aid for Shellfish poisonings and about shellfish preparation prior to cooking and consumption may be beneficially effective in the protection. Development of standard monitoring, determination and control units all around the seaside countries, adaptation of more specific determination techniques to more shellfish toxins and more specific identifications to more shellfish toxins with their hosts in higher sensitivities and lower costs are immediately required in minimizing public shellfish poisoning risk with an increasing possibility in a world globally warming.

Introduction

Sea water, a must for living and the world, is one of the most important media with its nutritional content for many various creatures which act as food to human and each other. Despite of its vitality on human and other living creatures, this media may also act as the transmitter of pathogen bacterial and viral agents of shellfish-associated illnesses such as Hepatitis A. Norwalk, *Vibrio parahaemolyticus*, *Vibrio vulnificus* and also toxins of planktonic algae which are dinoflagellates in most cases. These dinoflagellates which are the main causative agents with

their toxins use shellfish as host such as clams, mussels, oysters, scallops-cockles, starfish and crustaceans.

Several microscopic marine algae are notoriously poisonous to humans who consume them by the means of shellfish. These algae are tiny, single cell plants which look like plants on land, capture and use the sun's energy to grow. The growth of algae is an essential life process, as it is the first step in transferring solar energy into aquatic food. The huge variety algae are typically subject to annual cycles of growth and decay. These organisms thrive and multiply principally during spring and summer, in response to increase light intensity and salinity and also nutrients in ocean water. During the growth period or blooming, each single algae cell may replicate itself one million times in two to three weeks. During blooming, shallow seawater tends to become discolored by the sheer concentration of algae seeking the sunlight. This discoloration is a result of the various pigments which the plants use to trap sunlight that depends on the species of algae present. The water reflects pink, violet, orange, yellow, green, brown or red. Since red is the most common pigment, the phenomenon has come to be called 'red tide'. This 'red tide' is the main indicator for the dinoflagellates which elaborate and transmit the toxins through shellfish. Red tides are the most common in the waters of North America, Europe, Africa, Asia and Japan. The shellfish toxin associated poisonings are due to the contaminated shellfish consumption during red tide. Most species contributing to algal blooms are harmless but some species are poisonous to animals. Often, the algae themselves are unaffected as are the filter feeders like shellfish for whom micro-algae is the principal diet. However, these toxins are fatal for the carnivores including human. But in spite of the harmful dinoflagellates most common in cold waters, a few extreme identifications have also been made in warmer, neritic and tropic waters such as *Prorocentrum minimum*(Heil, Glibert and Fan, 2005), a bloom-forming dinoflagellate in Caspian, Black sea, Azov and Aral sea(Marasovic et al. 1998). Minor group of *Prorocentrum minimum* clones acts as sodium channel blockers, producing neurotoxic symptoms in mice, characterized with convulsions, spasms with pronounced palpitations and death within minutes.

Factors which effect toxin production and prognosis

Shellfish become toxic especially during blooming phase when environmental conditions are convenient. A bloom begins as a small population of toxic dinoflagellate cells in the lag phase or in the form of resting cysts, residing in the bottom sediment (Hall, 1982). Environmental conditions, required for the blooming are salinity, water temperature, nutrient density and sunlight. The higher water temperature, sufficient amount of nutrient with sunlight and salt trigger cysts germination in lag phase. During blooming, the growth is rapid with a tremendous increase in algae number which is called as log phase. Followed by blooming, as a result of CO_2 and nutritional wastes, the number tends to decline. This point is the start of the stationary phase, but also the highest algae number. The death cysts or the ones which are not bloomed settle down to the bottom or constitute a nutrient potential for it. High nutrient amount always trigger the blooming whereas low phosphorus concentration and low temperature increase toxicity. This may be probably because the cells continue saxitoxin production but reduced cell reproduction prevents toxin transfer to new produced cells(Anderson et al, 1990).The wind. the wave and the temperature are the other most important factors in the severity and transmission of the shellfish poisoning. The aerosol which is produced by the released toxin by the effect of the wind and wave can transfer the toxins and blooms to the far distances (Hortsman et al, 1991; Pierce et al. 2001).

Water current and wind acts importantly in the transfer of blooms to the far distances. The toxin free shellfish and dinoflagellate dominated beaches and shores can also be contaminated by toxic ones by the effect of water current and wind in the same direction while opposing wind and currents disperse the algae, decreasing the toxic density (Smayda and Shimizu, 1993). The resistance of clam is one of the important factors in severity and prognosis of shellfish poisonings which changes from species to species. Soft shell clams (e.g. *Mya arenaria*) and blue

ones from areas exposed to red tides are much more resist to saxitoxin than others and therefore accumulate toxins at greater rates than sensitive clams(Bricelj et al. 1990, 2005). Allowable limit enforced by the FDA for saxitoxin is 80 μg/100 g, whereas *Mytilus edulis*, a blue one in Alaska can accumulate 20.000 μg/100 g saxitoxin. The fish species which concentrates and transmit the dinoflagellate toxins is also important. For example, from these kinds of fishes which concentrates and transmits dinoflagellate toxins, the purple fish, can be considered to be one of the most unusual in appearance of all fishes in the ocean which belongs to tetraodontiformes order. It can be easily distinguished by their round plumb bodies which derives its name 'blow fish' or 'swell fish'. The spines in the body are unseen in the case they are calm, but during a predator approach, the spines immediately become visible as the fish causes its body to swell. The same fish when highly agitated fills a special sac inside its body with air or water. This inflation causes the fish to take on a balloon like appearance. They originate in the freshwaters of the Southeast Asia, but also they tolerate full saltwater. Tetradotoxin(TTX)is present in high concentrations in the liver of this fish with progressively decreasing amounts in ovaries, intestines and skin(Tsunenari, Uchimura and Kanda, 1980). Tetradotoxin poisoning can occur from the ingestion of a wide range of fish from tetraodontiformes, especially puffer fish (Meler and White, 1995).

One other red tide forming and classic causative organism for Shellfish poisoning especially in Florida, New Zealand, North Carolina and Gulf of Mexico is *Karenia brevis*, formerly known as *Gymnodinium breve* and *Ptycodiscus brevis*. The most dangerous point for *K. brevis* is its fragile structure which can easily be broken by the wave and release its toxins. Therefore, not only the location and the species but also toxin releasing capability due to the structural difference acts importantly in these kinds of poisonings.

Another important factor which affects the severity and prognosis of shellfish poisoning is the localization of toxin in shellfish. The gut and the intestinal tract of shellfish which have consumed toxic algae accumulate and store the toxins. As a result, the fish provided they are gutted in the usual manner, are not a threat nor ironically are scallops, a shellfish whose 'meats' the muscles that open and close its shell, are the only parts harvested for food. Therefore, gutting and cleaning process prior to cooking is an important factor which may act as significantly protective in reducing the severity of shellfish poisoning.

Difference in molecular structure of marine biotoxins is another important factor which effect toxin production and prognosis. A clear example is Saxitoxins which have a very close relation between the toxicity and their molecular structural difference. Saxitoxin molecules undergo chemical transformations that change from one molecular form to another. These transformations are two types. Epimerization, especially inspected in scallop and mussel, is the H and SO_3^-(sulfite) switch locations on number 11 carbon position which can decrease saxitoxin toxicity 11 times. If SO_3^- is separated by the effect of an acid on 21 carbon number, this type of transformation is called 'acid hydrolysis' and increases the saxitoxin toxicity 6 times. Therefore, not only the defference in molecular structure but also acid concentration of stomach may be a personal factor in determining the severity of PSP caused by saxitoxins. Saxitoxins are concentrated in the viscera and occasionally in the mantle and siphon of marine bivalves. Therefore, elimination of especially siphon as manually prior to cooking may decrease the severity of poisoning. This separation of siphon prior to cooking is deathly important in decreasing the toxicity since cooking does not completely destroy saxitoxins (Morse, 1977). Because Saxitoxin are detected in high ratios in viscera and gill (indicating recent poisoning since toxins move into the siphon after a period of time), two victims developed PSP because they had eaten clams which their siphons were not eliminated whereas others had no illness because of the separation of siphon prior to cooking in their meal.

Briefly, the factors determine the contamination risk and severity are as follows; the amount of toxic algae, the toxin content in each cysts, transformation rate to more or less toxic forms, the feeding rate, retention and excretion rate of marine biotoxin by shellfish, avoidance of toxic algae by the shellfish, gutting and cleaning process prior to cooking, difference in molecular structure and species of dinoflagellate, fish and shellfish.

Effect mechanisms, clinic, pathogenesis and treatment of marine biotoxins:

TTX, ST and domoic acid are selective blockers of voltage-sensitive sodium channels and prevent conduction in motor and sensory nerves by blocking sodium channels at the nodes of Ranvier (Meler and White, 1995).

TTX poisoning occurs from southern New South Wales to northern Queensland in shallow coastal waters and estuaries and with a potential in many coastal regions of Australia. Symptoms generally occur 10-45 minutes after eating the puffer fish and begin with numbness and tingling around the mouth, salivation, nausea and vomiting. Symptoms may progress to paralyze, loss of consciousness and respiratory failure and can lead to death. Vomiting should be induced if the person is awake and alert within 3 hours of poisoning. Artificial respiration (sometimes with cardiopulmonary resuscitation) and turning onside may keep the person alive until medical attention. A new data has just come from Landsberg et al(2006), indicating a new emerging public health threat, *Pyrodinium bahamense* (dinoflagellate) as the putative toxin source which causes fatal Paralytic Shellfish Poisoning(PSP) in Pacific, but not known to be toxic in western Atlantic. It causes a syndrome which can be called as 'Saxitoxin Puffer Fish Poisoning (SPFP) which is traditionally associated with tetrodotoxin. (Landsberg et al, 2006).

Brevetoxins controversially, open voltage gated sodium (+Na)ion channels in cell membranes, leading to uncontrolled Na+ influx into the cell(Purkerson et al. 1999) which alters the membrane properties of excitable cell that enhances the inward flow of Na+ into the cell. Brevetoxins can also pass through cell membranes including blood brain barrier, mucosa and skin (Apland et al. 1993). These toxins release acetylcholine followed by Na+ influx which causes to the smooth tracheal muscle contraction as well as massive mast cell degranulation. The histamine release of degranulated mast cells is the main causative factor of the asthma which is inspected during Brevetoxin Shellfish Poisoning. Lipid solubility of the concerned toxins indicates to the possibility of the brain damage which is sometimes accompanied with brevetoxications (Apland et al. 1993). Depolarization of the nerve cells by brevetoxins disrupts respiratory, digestive and cardiac function. Therefore, brevetoxins are also cardio and hepatotoxins (Rodriguez-Rodriguez and Maldonado, 1996). Fish, birds and mammals are susceptible to the brevetoxins (Bossart et al. 1998). The red tide toxin aerosol inhalation and ingestion from contaminated seawater caused 149 manatee deaths in the Gulf of Mexico. Pulmonary hemorrhage and oedema, meningitis(inflammation of brain membranes) , hemolytic anemia and catarrhal rhinitis(inflammation of the nose or nasal membranes) were the main post mortem clinic view of the manatees, as indicating to the respiratory tract, liver and kidneys as the primary targets of brevetoxins in mammalian metabolism (Trainer and Baden, 1999). It sometimes needs for respiratory support whereas brevetoxin aerosol inhalation causes coughing and burning in nose and throat, rhinorrhea (persistent watery mucus discharge from the nose) and broncoconstriction (Temple, 1995). The respiratory disorder is usually rapidly reversible by living the beach or contaminated water (Steidinger and Baden, 1984). Whether the inhalation of aerosolized brevetoxins can result in neurological or immunologic systems apart from upper respiratory is not known and needs further studies (Fleming et al, 2001; Benson et al, 1999). Mortality is low. (Martin et al. 1993).The severity of pulmonary, gastrointestinal and neurological symptoms during Brevetoxification depends on the toxin concentration in aerosol. Broncocontractions can be blocked by verapamil, atropine and tetrodotoxin(Jeglitsch et al.

1998). Histamine antagonists such as chlorpheniramine (Abraham and Baden, 2001) and Mannitol's early intravenous administration can be effective in early stages of brevetoxifications by the excretion of Na+ ions without their reabsorbtion (Blythe et al, 2001; Mattei et al, 1999). Monoclonal antibodies and antidotes are other protective treatments (Templeton et al, 1989a). Cromolyn and chlorpheniramine which prevent the immune system's mast cells from rupturing when they come into contact with an allergen stabilize these mast cells and prevent them from bursting and releasing histamines are also used in NSP which is occasionally caused by brevetoxins. Others which are commonly used in treatment of Brevetoxifications are calcium channel blocker verapamil, sodium channel blocker tetrodotoxin, Na+influx blocker atropin and acethylcholine blocker beta 2-agonists(Trainer et al. 1991; Singer et al, 1998; Watanabe et al, 1988). Canning does not decrease brevetoxin concentration in bivalves (Viviani, 1992). Successful ozone-assisted depuration of red tide contaminated shellfish, both killing the organism and inactivating the toxin, has been reported whereas ultraviolet and chlor were unsuccessful (Baden et al, 1995; Fletcher et al, 1998; Roderick, 1997). Laboratory equipment should be decontaminated by soaking in 0.1 N NaOH solutions for 10 min. prior to the combustion in chamber at 500 °C. Steam autoclaving is not successful in decontamination pf brevetoxins. The brevetoxins produced by *K. brevis* and other breve dinoflagellates are tasteless, odorless, heat and acid stable. These toxins can not be easily removed by food preparation procedures such as heat and acidity (Baden and Trainer, 1993). The brevetoxin concentrations in contaminated clams have been reported to be 78-120 μg/mg (Poli et al. 2000). Main clinic view of NSP is a sum of gastrointestinal and neurological abnormalities such as: nausea, diarrhea, abdominal pain, paresthesia (abnormal skin sensations usually associated with peripheral nerve damage), vertigo (dizziness), incoordination, bradycardia(unusually slow heartbeat), dilated pupils, convulsions and headache.

Another shellfish poisoning is Paralytic Shellfish Poisoning (PSP). The causative agents are *Alexandrium* genus contaminated by the means of bivalves such as clams and mussels. There are at least 21 molecular forms of PSP toxins which are termed 'saxitoxin'. Saxitoxin derives its name from the butter clam, *Saxidomus giganteus*, where saxitoxins were first originally extracted and identified. All saxitoxins are neurotoxins that block the movement of sodium through nerve cell membranes(like in tetradotoxins and domoic acid but opposite to brevetoxins which do not block, controversially enhance Na influx) which results in numbness, paralysis, disorientation which forms paralytic syndrome of PSP. The toxicity of PSP toxins is 1000 times greater than cyanide. In 1987, these toxins had cause to deaths of whales. The killing of whales by PSP continued 50 years. 3 people died by the end of 1987 with the symptoms including dizziness, diarrhea, vomiting, respiratory distress and eye irritation while over a hundred had been severely incapacitated by PSP. The causative agents, saxitoxins, are one of the most toxic non-protein substances ever known. Only a single dose of 0.2 mg is fatal. The most common causative algal dinoflagellate species of PSP are *Alexandrium tamarense*, *Gymnodinium catenatum* and *Pyrodinium bahamense* which are fed and ingested by filter feeding mollusks where also the concerned toxins are concentrated in. Saxitoxin, is also 1000 times more toxic than synthetic nerve gas, sarin. It is not surprising that in 1950s, the CIA began experimenting it, reportedly using it in suicide capsules(is a pill, which military personnel sometimes carry to prevent secrets from falling into enemy hands during capture, implanting it under tooth so that, biting down hard was going to cause toxin to be released) provided to its agents, notably U-2 pilots. In 1970, President Richard Nixon ordered CIA to destroy entire saxitoxin stock, as part of the US commitment in accordance with the United Nations agreement on biologic weapons. Ricin and Saxitoxin are the only two natural toxins which are presently reported. Ricin is produced by *Ricinus communis*(castor bean) and can be transmitted through inhalation, consuming, drinking the contaminated water (like saxitoxins) or even contact with plant root. Georgi Markov, a Bulgarian writer, had been killed with an attack by an umbrella in 1978. The umbrella had been rigged to inject ricin under Markov's skin that only a pinful(~500 μg)is totally enough to kill a

human. The concerned toxin kills by blocking RNA and protein synthesis. The main problem at this point is the threat potential of these biotoxins which may be used as biologic weapons if they are not strictly controlled and monitored. Saxitoxins like ricin cause paralyze by blocking Na+ channels, but have no effect on K and Ca channels or the flux of Cl-. PSP is the most commonly reported marine biotoxification with ciguatera and scombroid poisoning. *Protogonyaulax* and *Alexandrium* dinoflagellates which are consumed by bivalve filters such as oyster and mollusk are the initial transmitters of PSP toxins. A severe poisoning can occur and led to death from respiratory paralysis in 2-24 h. Crabs and also some fresh water *Cyanobacteria* can also appear as having these toxins (Novak, 1998). The main clinical symptoms of PSP (and saxitoxifications) are acute onset of paresthesia, nausea, vomiting, severe headache, dysarthria (impaired articulatory ability resulting from defects in the peripheral motor nerves), dysphagia(difficulty in swallowing) and ataxia(inability to coordinate muscular movement). Hyper tension such as 172/110 mmHg, pulse 83, pink skin and mucous membranes accompanied with dysarthria and dysphasia, cyanosis in severe cases requiring mask ventilation and endotracheal tube amy also be inspected in clinic view. Lymph nodules are normal, there is no abnormality as related to cardiac tissue, lung and abdomen except diluted pupils and areflexia (absence of reflex)(Gessner, Middaugh and Doucette, 1997). 2 glasses of water with 3 tablespoons of activated charcoal is given in the emergent first aid to the person in PSP(and saxitoxifications) which sometimes may be supplied by CPR and turning onside.

'Domoic acid Shellfish Poisoning' (DSP) or 'Amnesic Shellfish Poisoning' (ASP) can be characterized as a neurotoxicity. Domoic acid is produced by *Pseudo nitzschia,* a unicellular diatom (algae) shows its toxicity not only in humans but also in several hundred animals (Moss, 2001). In this toxicity, the clinic is the sum of gastrointestinal and neurological disorders which defects especially amygdala and the hippocampus (Teitelbaum et al, 1990). Domoic acid toxicity is because of the reduction in voltage dependent Mg2+ block at the neuromuscular domoic acid receptors followed by the activation of non neuromuscular domoic acid receptors by domoic acid (Novelli et al, 1992). Mussels, clams and oysters can be the transmitters of domoic acid. Domoic acid is not destroyed by cooking or freezing. Because there is no antidode for ASP, prevention is the best protection. Domoic acid causes neuropathy (defect of neuron) and axonopathy (defect of axon). Domoic acid or in other words *Pseudo nitzschia* diatom toxins also decrease glucose metabolism in the medial temporal lobes. Neuronal necrosis and loss in the hippocampus are the main factors leading to death (Teitlebaum et al, 1990). Confusion and disorientation are also complicated in all ASP disorders. Other symptoms are vomiting, nausea, diarrhea and abdominal cramps, dizziness, confusion, motor weakness, loss of short term memory, cardiac arhytmias, and headache and in severe cases coma and death. Among symptoms, short term memory loss is permanent which also gives the name 'Amnesic'. Relative preservation of intellect and higher cortical function appears to distinguish ASP from Alzheimer and the absence of confabulation is not typical of Korsakoff's syndrome (an alcohol toxicity on neurons which closely related to the B vitamin deficiency due to alcoholism which is characterized by neuropathy). Polyneuritis(inflammation of several nerves), delirium(overly excited state), anxiety, fear, depression, confusion, insomnia, painful extremities, pain over the long nerves are the other clinic symptoms but confabulation(ability to talk and conversation) is exist.

An important question. Which one is the causative agent of Shellfish Poisoning as toxin producer? Dinoflagellate or bacteria?

Hall(1982)confirmed dinoflagellate connection of high amount resting cysts of *Alexandrium cantenella* with bacteria. The later findings indicated to the higher levels of the concerned toxins of some geographical strains while other strains of the same algae produce fewer toxins. Silva and Sousa(1981) made a remarkable discovery by transforming a non toxic dinoflagellate strain to a toxin producer by simply inoculating the non toxic strain with a bacterium, *Pseudomonas*

spp., isolated from a toxin producing dinoflagellate. Here the question is; which organism, dinoflagellate or bacterium is responsible from saxitoxin production.

Dinoflagellates routinely harbor intracellular bacteria (Bold and Wynn, 1979) that also indicates to the same question. Most of the scientists first claimed that it was not just because of the bacteria since most of them were psychologists expressing the bacteria do not produce toxins. They only induce the algae to synthesize saxitoxins. But the saxitoxin production by bacteria, followed by the separation of it from algae supported strongly the saxitoxin production capability or probability of bacterial saxitoxin production(Kodama et al, 1988, 1990). Later, *Cyanobacteria*(formerly known as blue-green algae) which are fresh water bacteria flora, were proven to produce saxitoxins(Silva and Sousa, 1981). Another question appeared. Do only fresh water bacteria produce saxitoxins.? Presently, we face with similar important questions as related to the bacteria-algae connection such as; does the dinoflagellate or bacteria produce saxitoxin?, if bacteria is the one to produce, how they are capable to produce large amounts of STX, whereas the same bacterial saxitoxin production is very little in laboratory conditions?. Are there genetic factors such as 'trans-kingdom sex', that transfer the genes, which produce saxitoxins to the algae from bacteria? Meanwhile, the bacterial composition of toxic dinoflagellate cultures is becoming increasingly known (Graben et al, 2001). Specific bacteria may be stimulated by algal exudates (Bell and Mitchell, 1972 and Shapiro et al, 1998). Also a small percentage of bacteria are found to be attached to live or dead algal cells including harmful algae blooms (Albright et al. 1986; Smith et al. 1995; Bidle and Azam, 2001; Rausch de Traubenberg et al, 1995; Watson et al. 1996; Lewis et al. 2001; Alavi et al. 2001; Kaczmarska et al. 2001, Biegala et al. 2002 and Simon et al. 2002). Although it is known that some bacteria attached to particles are physiologically more active than free-living bacteria(Smith et al. 1995), their significance to algal physiology and bloom dynamics is still not clear. Bacteria are also cited as important regulators of algal toxicity (Tosteson et al. 1989, Bates et al. 1995 and Doucette at al. 1998). However, there is conflicting evidence as to whether bacteria increases, decreases or have no effect on the toxicity of dinoflagellates that PSP toxins (Dantzer and Levin, 1997; Hold et al, 2001b, Simon et al. 2002). Gallacher and Smith (1999) reviewed the literature on the ability of bacteria to produce sodium channel blockers and saxitoxin. Cordova et al. (2002) reported the intracellular bacteria from *Alexandrium catenella* which can produce small amounts of saxitoxin. Specific bacterial communities are associated with toxic and non toxic dinoflagellates(Hold et al, 2001a) and axenic cultures of *Alexandrium catenella* produce less toxin than xenic cultures(Uribe and Espejo, 2003). The role of bacteria in Domoic acid production remains an active area of investigation. Both axenic and xenic cultures produce domoic acid, but in the absence of bacteria, *Pseudo nitzscia* cultures produce 8-38 times less Domoic acid than xenic cultures (Douglas and Bates, 1992; Douglas et al. 1993, Bates et al. 1995). The reintroduction of bacteria to axenic cultures caused a 2-115 fold enhancement of Domoic acid production relative to axenic cultures (Bates et al. 1995).

Withdrawal time of shellfish toxins (Depuration)

After a period, the shellfish toxins are cleared by shellfish which is called 'depuration'. The time required for depuration changes between 20-50 days, depends on genetic and environmental attributes of shellfish.

Type of the tissue, species, food availability and the media, the level of non toxic algae, particulate concentration, temperature, salinity and nutrient content of water media are the main factors which determine the degree of depuration. The toxins in the gastrointestinal tract are eliminated much more readily than the toxins bound in tissues which explain the importance of the type of the tissue. Another important factor is the species. Factors which effect toxin production and prognosis mentioned above also act importantly in depuration. Higher tolerance

of blue clams such as *Mytilus edulis* to STX with one week to six month retention time is a good example to show the importance of the species when toxicity and depuration concerned. Another important factor for depuration is the media. DSP toxins drop to acceptable levels more quickly in the aquaculture pond than natural media indicating to the importance of media and food availability in the media (Hallegraeff et al, 1995). In the Netherlands, mussel marine toxins disappeared after 4 weeks at 14-15 °C water temperature (Hungerford and Wekell, 1992) while they decreased from 7.2 to 1.8 mg/g only at 1.4-3 °C in Sweden coast(Edebo et al, 1988b) which manifest the importance of heat in depuration..

Can surface water temperature increase by global warming be a new threat of shellfish poisonings?

In the last decade, water surface temperatures increased 1 to 3 °C due to the global warming. When the same longitude and latitude intersections, for example 60N-30W, 30N-150W, 15S-120W and 39S-90W are accidentally selected, these 1 to 3 °C increases in surface water temperature(NODC,) which belong to 1998 and 2005 years are striking. It seems definitely possible. It is not very difficult to predict the hazardous effect of water temperature increase by global warming when the data as related to the relation of heat and dinoflagellate multiplication and toxin production is evaluated. One of the clear examples of this relation is *Gymnodinium catenatum* at 20-25 °C whereas it can not grow at 10 °C and lower than 10 °C (Nagasoa et al. 2006). Also Bravo et al.(1994) observed a high rate of growth of the same organism at 22-28 °C in Spain coast waters but little or no germination in the cold waters of northwest Spain. Similarly, from 60 different combinations of temperature (10-30 °C) and salinity (10-40), under saturated irradiance, *Cochlodinium polykrikoides*, an harmful red-tide dinoflagellate, exhibited maximum growth rate at 25 °C with a salinity of 34. The same organism could grow in all temperatures between 15-30 °C in salinities from 20 to 36 but could not grow at 10 °C or lower than 10 °C without respect to salinity (Kim et al. 2004). *Alexandrium monilatum* was another dinoflagellate which grew faster up to 31 °C (Juhl, 2005). Slight increases of surface water temperatures especially from 1998 to 2005 as 1 to 3 °C means 6 to 9 °C or even more temperature increases in water surfaces in the next three decades. Volume increase of Gulf stream by the effect of global warming in another important factor which increases water surface temperatures. A brief explanation and recall of Gulf Stream occurrence will be beneficial to be able to realize the potential threats of global warming from the point of shellfish poisoning. Melting of huge amounts of ice in the Arctic region increases fresh water amount on surface which creates a warm, less dense stream. This stream begins from just below of Arctic region and north Atlantic, circulates through south Atlantic, west Europe, north Africa, coasts of India and China, north Australia and New Zealand and reaches to Arctic region. Through this circulation, by the effect of the UV, gases and radiation and also heat, significant amount of water is evaporated and becomes more salty and dense. This second stream created due to its high density and salt content circulates in deep not on surface. This deep stream, as cold and in high density makes its circulation through north Australia, coasts of China and India, north Africa, west Europe and reaches back to the Arctic region to gain more fresh water due to the ice melting in Arctic. This cycle continues endlessly as long as ice in Arctic region melts. The threat at this point is the global warming which triggers temperature increases in Arctic regions, resulting in higher volume of fresh water less salty and less dense, in other words a water volume increase of Gulf Stream. This increase of temperature on water surface through gulf stream may stimulate dinoflagellate growth which tends to grow on surface in higher temperatures such as ; orders of *Gonyaulacales*, *Gymnodiniales*, *Peridinales* with *Pyrodinium bahamense* var. *compressum* and Polysphaeridium zoharyii of southeast Asian waters(Furio et al. 2006) and the orders of *Littorinimorpha*(e.g. *Bembicum nanum* and *Polinices* sp.) and *Basammatophora*(e.g. *Siphonaria zelandica*)(Przeslawski and Benokendorff, K. 2005). Briefly we will be able to face the risk of some types of surface harmful algal boolms stimulated by increased gulf stream

volume by the effect of global warming, a theory but always a great possibility, which has got a great potency to turn into reality in a very near future. The second threat may be the cold stream's (in deep mentioned above) stimulating effect on some algal blooms which requires cold temperatures in deep which is also more nutritious such as;harmful red alga, *Gracilaria tikvahiae* (Maehira et al. 2004) or tropical reef alga *Halimeda tuna* on Conch Reef of Florida(Smith et al. 2004). Just because of these two mentioned risks due to the global warming, in the depth and on surface, because of the stimulation of surface and deep algal blooming due to the global warming, shellfish poisoning will matter more than ever for the next decades with higher mortalities.
This possibility in the case of absence of the urgent protective actions against global warming may bring out the risk of excess growth of the dinoflagellates mentioned above which their growths are stimulated by higher temperatures. We face with an important milestone that first, we should be conscious of this stage where the standardization with uniform monitoring program is immediately required but before, the improvement of a standard marine bioassay in higher sensitivity with lower cost and timely computerized communication of seaside countries should be constituted without wasting any more time. This strategy should be followed by food hygienists, food technologists, food scientists, marine scientists and educators in accordance with the other protective actions in other platforms.

What should be done if it matters and will matter more than ever in the future:
In fact, preventive measures are very limited and restricted because toxic dinoflagellates are various and can easily contaminate far and open sea by the effect of wind and wave. One common problem is that certain algal species which have never occurred in a certain area may suddenly appear and then rapidly cause problems. Some countries monitor the presence of only one or two algal species. Except Italy, other countries close their harvest areas only when the toxins have been detected in the shellfish. However, like Italy's approach, the shellfish areas should be closed when the number of cells of certain algal species exceeds certain concentrations according to the type of species (Hallegraeff et al. 1995). The principal strategy in prevention of marine intoxication should be the effective monitoring so that contaminated products do not reach to the market. Unfortunately, this may not yield a complete protection since weekly sampling may be insufficient for maximum protection. Also several factors complicate efficient monitoring like; 1. Cell numbers of toxin producing algae needed to produce toxic mussels vary considerably 2. Time period of toxicity varies with region and episode 3. Implicated toxins and algae may be different in different regions 4. Simultaneous presence of marine toxins, especially DSP and PSP toxins complicates monitoring 5. Toxicity of contaminated mussels can vary several folds with different depths, even at the same sampling location and 6. Not only bivalves but also other seafood like oysters might also be contaminated although at a lower level.

As in the highlight of the risks mentioned above, the protective actions which should be taken immediately are as follows; 1. A standardized, uniform marine bioassay method in higher sensitivity with less cost should be modified or decided and should gain a using platform by all seaside countries 2. A well computing communication should be constructed among the same seaside countries to be able to warn each other timely in the case of harmful alga blooming and contamination risk and 3.The country which detects harmful algae blooming and contamination should close and take its harvest area under quarantine and inform it immediately to the other seaside countries by using computing system. These three protective actions, as easily guessed, will require a great organization, fund and labor but should be created without wasting any more time to be able to prevent the potential hazards of harmful algae risk in the future.

Destruction of marine biotoxins:

Transfer of shellfish to waters free of toxic organisms for self-depuration, vertical displacement of mussels in the water column as a means of minimizing toxin accumulation, ozone treatment, salinity stress, electric shock, reduced pH, chlorination and cooking, all appeared to be unsafe, too slow and economically unfeasible, yielding products unacceptable in appearance and taste(Hallegraeff et al. 1995). The most effective method for toxin destruction seems only rigorous boiling at 100 °C as long as 163 minutes. The final research is about 90% toxin elimination by CO_2 and acetic acid super critical atmosphere creation (Gonzales et al. 2002).

Marine Bioassays and their comparison in Diagnosis

The Mouse Assay is the most commonly and initial one to detect marine toxins in many countries presently.. In this assay, shellfish extract, treated with acetone and toluen 60, is injected intraperitoneally to a mouse in a weight of about 20 g(Yasumoto et al, 1978). 1 Mouse Unit (MU) is defined as the smallest shellfish toxin which kills this weight of animal in 24-48 h. The toxicity of the concerned shellfish toxin is determined from the smallest dose which can kill two or more animal in a group of three within 24 h. Unfortunately this test has got advantages which are mainly; lack of specificity, subjectivity in death time of animals, maintaining and killing of laboratory animals and interference by other lipids which requires an additional hexane washing treatment. Unfortunately hexane's application may decrease domoic acid, results in significant loss of this toxin which also decreases the sensitivity of the concerned assay.(Fernandez et al, 1996). Therefore it is worthy to note that hexane treatment should be avoided in the cases of domoic acid determinations. Sackling Mouse Bioassay is a little more sensitive as compared to Mouse Bioassay, which requires the administration of toxin as intragastrically and the measurment of the fluid retention in the intestines followed by toxin administration. But the fluid amount detection due to the toxicity of injected toxin is very difficult and only diarrhoea causing toxins can be detected by this method (Hallegraeff et al, 1995). Rat Bioassay uses the inspection of diarrhoea formation due to the toxin administration during 16 h but it can only detect Paralytic toxins (Hallegraeff et al, 1995) causing diarrhoea. The Assay of Daphnia magna, a freshwater invertebrate, is more sensitive than Mouse bioassay but its application is restricted to only Okadaic acid (Vernoux et al, 1994). Another assay which is called 'intestinal loop assay' uses the rat or mouse and the administration of the toxin. The ratio of the fluid accumulated as ml to the intestinal loop as cm when becomes greater than 1.0, the result indicates to the toxin existence. This assay is also restricted to only DSP toxins(Hungerford, 1992). Cytotoxicity assay is based on the morphological changes in rat hepatocytes (Aune et al, 1991) but it is limited, and only capable to detect diarrhoeic and nondiarrhoeic DSP toxins and also very complex which may yield confusing results. in the case of many different algal toxin existence since different toxins cause different marphological changes in rat hepatocytes. Mouse Neuroblastoma Cell Assay (MNCA) is based on morphological change of neuroblastoma cells. Mouse neuroblastoma cells swell and lyse by the effect of veratridine and quabain which enhance Na influx. PSP toxins especially saxitoxins block sodium channels and therefore inhibit these two compounds and remain cells normal. The fraction of the normal cells protected from the action of veratridine and quabain is in direct proportion of STX. The main problem about this test is its narrow application range that can only be applied to PSP including STX (Truman and Lake 1996). Heamolysis assay differs from neuroblastoma which uses red blood cells of red tilapia (*Sarotherodon mossambicus*). Veratridine and quabain change cell membrane permeability by causing Na influx whereas STX inhibits. The fraction of normal red blood cells are in direct proportion of STX(Shimojo and Iwaoka 2000). But this test, also like neuroblastoma cell assay, could not be applied to other toxins except STX. Cheun et al.(1998) modified this method to a Na electrode covered with a membrane. The Na transfer in the absence of Na channel blocker from the internal to the external side of the membrane is in direct proportion of STX. It is also only applied STX and other PSP toxins and still waiting to be applied for the detection of other biotoxins. Recent promising brevetoxin detection methods include HPLC, HPLC-MS,

fluorescence, ELISA(based on goat anti-brevetoxin) and radioimmunoassay (Trainer and Baden, 1991)which can be commonly applied to contaminated seawater, air and contaminated shellfish(Naar et al, 2002). For the same toxins the latest qualitative detection uses an immunohistochemical stain for the toxins within macrophages and lymphocytes in nasal mucosa, lung and other tissues (Bossart et al, 1998). No tests are available in human fluids whereas HPLC-MS and ELISA in urine are promising (Naar et al, 2002). uses rabbit antibody and STX and PSP toxin antigen binding with a good sensitivity(which is also confirmed by Mouse bioassay. Unfortunately this test has lack of response to other toxins and therefore still remains very limited. LC (Liquid Chromatography is based on separation of toxins by ion interaction chromatography and use of a post column reactor that oxidizes the column effluent to produce readily detectable amounts. In this technique calibration is essential by using PSP toxin standards which is not easy many times since there are significantly important differences among STX and other PSP toxins. But another problem about this method is the limited application which includes only STX and some PTX. Slab electrophoresis uses gel and paper electrophoresis (Thibault et al, 1991), allows rapid screening of many toxins and very promising for the near future in the mean time. Capillary electrophoresis uses two silica capillary in a place of electrophoretic gel. Between two capillaries a column is exist which toxins are introduced from one end and pass the column from one capillary to another followed by high voltage administration to the column. During its migrate, toxin can be detected by UV or fluorescence. This is one of the few methods which is suggested by many scientists considerably promising whereas only problem appears as very high detection limits which will limit its application in such monitoring programs. LC-MS(Liquid Chromotography and Mass spectrometry has got the same disadvantage of LC, the standards, which are required for many toxins than STX and PSP toxins. Therefore this method also should be studied well to gain certified standards of toxins. Hippocampal slice assay (Kerr et al.1999) which is based on morphological changes in hippocampal tissue of mouse is also limited, capable to detect mainly PSP toxins. The synaptosome binding assay is a competitive binding assay in which radiolabelled NSP toxin or its derivative compete with unlabelled NSP toxin for a given number of available receptor sites in a preparation of rat brain synaptosomes. The present reduction in radilabelled NSP binding, is directly proportional to the amount of unlabelled toxin present in an unknown sample (Poli et al, 1986).

Briefly, we face with serious problems as related to the detection marine origin toxins rapidly and sensitively with a low cost. Firstly; with its subjectivity in death time, killing of laboratory animals and interference with lipids, Suckling Mouse Bioassay with it's difficult procedure, do not seem as the method which can be applied commonly in marine biotoxin determination. ELISA, heamolysis, Neuroblastoma cell assay, cytotoxicity assay, Daphnia magna, Hipocampal slice, Suckling Mouse and, synaptosome binding assays can only detect one or some group of toxins whereas LC and LC_MS have lack of standards of a wide range of marine biotoxins. Although Capillary electrophoresis is the most promising one for the near future, needs urgent improvement to lower high detection limits. In the overall evaluation of the concerned biotoxin determination methods, there is no perfect bioassay to determine marine biotoxins. We should consider two important factors. First, all of these assays have got deficiencies as related to standards, narrow range of application, limited to very few biotoxins, high cost, complexity. That is why, it is urgently needed to fulfill these deficiencies and manifest a marine bioassay in high sensitivity and low cost. Secondly, all countries especially Australia, west costs of United States, Mexican gulf, Venezuela and Argentina, Mediterranean countries, China, Japan and India should modify, manifest a uniform assay in high sensitivity and low cost, accept and apply it immediately. It is my point of view that all these countries should have got a forceful monitoring program which uses both Mouse bioassay and LC or LC-MS (as one for screening and other for quantity detection) until a new well modified and improved bioassay manifestation. The most important point here is the entrance of bioassay results and communication of these

countries by computing to warn each other and give an immediate alarm in the case of a severe marine origin biotoxin terror.

As mentioned above, monitoring and assay is one of the greatest problem we face about biotoxin thread which also will continue to be greater because of global warming. Ireland's programs include weekly mouse bioassay testing and a web based information system which is still being developed to increase access to the information (McMahon et al. 2001). Turkey has no further information and regulation except mouse bioassay(Fernandez, 2000). The Republic of Korea determines weekly by using LC(APEC, 1997) and Mouse bioassay, also has got a monitoring system. China has got two regular monitoring programs for the north and the south of the country with weekly plankton and shellfish monitoring (APEC, 1997). Uruguay has national monitoring programs (Fernandez, 2000), Venezuela, like Turkey, has got only mouse bioassay (Fernandez, 2000). Chile has got 2 monitoring programs with mouse bioassay, LC and LC-MS (Fernandez, 2000), Brazil does not have a national monitoring program (Ferrari, 2001) whereas USA and Argentina have (APEC, 1997). Australia has got its own regulation and monitoring, while New Zealand has got a monitoring program which combines regular shellfish testing and phytoplankton monitoring (Busby and Seamer, 2001). As clearly seen, there is no standardization among the concerned seaside countries from the point of marine toxin detection and monitoring. A web based information plus LC, LC-MS which does not need a further mouse bioassay (McNabb and Holland (2001) which is being performed by Ireland presently can be the present monitoring and alarm system for other mentioned countries. Its acceptance and maintenance by all these countries may also eliminate standardization problem in biotoxin determination and monitoring.

Conclusion

We face with a critic stage, climacteric and susceptive too many risks including the risk of shellfish poisoning which may increase in a very near future by the effect of global warming. Main adverse effect of global warming may be its stimulating on deep and surface toxin producing dinoflagellates by increasing surface water temperatures and decreasing deep water temperatures day by day. The harmful effects which may be defined as the sum of pathogenesis on neurons, pulmonary-upper respiratory and digestive system is very net and clear as in the highlight of present data. Unfortunately, we still have neither uniform, standardized bioassay which has high sensitivity in low cost nor control nor monitoring systems among seaside countries. Controlling' and 'monitoring of the harvest areas is still deficient for many countries. New shellfish poison detection and monitoring units with international communication based on computing are immediately required with a proper standardization which weekly monitor algae in harvest area. This seems the only protection which we may have presently whereas bacteria-algae interrelation, new methods for depuration and elimination of shellfish toxins need urgent studies which should be performed immediately.

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ARBEITSGEBIET LEBENSMITTELHYGIENE

48. ARBEITSTAGUNG

des Arbeitsgebietes Lebensmittelhygiene

– Dreiländertagung –

zusammen mit der
Sektion Lebensmittel tierischer Herkunft in der
Österreichischen Gesellschaft der Tierärzte

und der

Schweizerischen Tierärztlichen Vereinigung für
Lebensmittelsicherheit

In diesem Jahr wird das Programm zusammen mit der Akademie der Österreichischen Tierärztekammer (VETAK), Wien, gestaltet

vom 25. bis 28. September 2007
in Garmisch-Partenkirchen
Kongresshaus

Tagungsprogramm

Beginn **13.30 Uhr**
Kongresshaus Garmisch-Partenkirchen
Saal „Werdenfels"

Musikalischer Auftakt
Ensemble „Blechvet" der Tierärztlichen Fakultät
der Ludwig-Maximilians-Universität München

Begrüßung durch Herrn **Univ.-Prof. Dr. Dr. h.c. A. Stolle**
München

Musikalische Einlage

Eröffnung

Grußworte

Musikalische Einlage

Festvortrag
„Was drauf steht ist auch drinnen"
W. Lampert, Initiator von „Zurück zum Ursprung" (Wien/A)

Eröffnung der Industrieausstellung

Musikalischer Ausklang

Kaffeepause **15.30 Uhr**

Saal „Werdenfels" - Lebensmittelrecht

Kobelt, H. (Bonn) Informationen zum aktuellen Stand der Weiterentwicklung des EG- und nationalen Lebensmittelrechts	**15.50 Uhr**
Stetter, B. (Stuttgart) Zulassung von Betrieben	**16.05 Uhr**
Diskussion	**16.20 Uhr**
Löpfe, J. (Wallisellen/CH) Die Notwendigkeit der Nachvollziehbarkeit tierärztlicher Tätigkeit in der Primärproduktion aus Sicht der Lebensmittelindustrie	**16.30 Uhr**
Kulow, W. (Recklinghausen) Leitlinien für eine gute Verfahrenspraxis – Bedeutung und Anwendung im neuen Hygienerecht	**16.45 Uhr**
Werner, G. (Bonn) Die Health Claims-Verordnung und ihre Anwendung	**17.00 Uhr**
Diskussion	**17.15 Uhr**
Fehlhaber, K., G. Hildebrandt (Leipzig, Berlin) Die neue Tierärztliche Approbationsverordnung aus Sicht der Ausbildungsqualität in den Lebensmittelfächern	**17.30 Uhr**
Fischer, M., K. Fehlhaber (Leipzig) Studie über die Ausbildung von Tierärzten in den Lebensmittelfächern: ein europäischer Vergleich	**17.45 Uhr**
Diskussion	**18.00 Uhr**
Ende	**18.10 Uhr**

Saal „Werdenfels" - Lebensmittelhygiene

David, H. (Düsseldorf) **8.30 Uhr**
Der „Smiley" – ein neuer Weg der Lebensmittel-Überwachung in Nordrhein-Westfalen?

Johne, R., S. Kaci, K. Nöckler (Berlin) **9.00 Uhr**
Hepatitis E – eine über Lebensmittel übertragbare virale Zoonose in Deutschland?

Moehl, K., A.S. Manopas, G. Hildebrandt (Berlin) **9.15 Uhr**
Mikrobiologischer Status und Nachweis von Noroviren in frischen Obstsäften und -salaten

Diskussion **9.30 Uhr**

Pichner, R., C. Scherbel, M. Gareis (Kulmbach) **9.45 Uhr**
Untersuchung zur Stabilität des Prion-Proteins im bovinen Gastrointrestinaltrakt

Gebert, R., A. Didier, R. Dietrich, E. Märtlbauer (München) **10.00 Uhr**
Vergleichende Untersuchung zur PrP^{c}-Expression in Euter und der Milch bei kleinen Wiederkäuern

Didier, A., R. Gebert, R. Dietrich, E. Märtlbauer (München) **10.15 Uhr**
Vorkommen von Prionen in Colostrum und Milch BSE- und Scrapie-infizierter Schafe

Diskussion **10.30 Uhr**

Kaffeepause **10.45 Uhr**

Saal „Werdenfels" - Lebensmittelhygiene

Hamedy, A., T. Alter, K. Fehlhaber (Leipzig) **11.05 Uhr**
Qualitative und quantitative Belastung von Putenkarkassen (Halshaut) und Putenfleisch mit *Campylobacter* spp.

Wadl, M., G. Flekna, I. Hein, L. Thompson, **11.15 Uhr**
J. Slaghuis, J. Köfer, M. Wagner (Wien/A, Newport/GB, Darmstadt)
Entwicklung einer Schnelltestmethode zum direkten Nachweis von *Campylobacter* spp. aus Hühnerkot

Mayr, A., S. Lick, A. Anderson, C. Günster, **11.25 Uhr**
D. Thärigen, R. Zucker, I. Huber (Oberschleißheim)
Untersuchungen von Lebensmittels auf *Campylobacter jejuni, coli* und *lari* mittels Multiplex real-time PCR parallel zur Routinediagnostik

Diskussion **11.35 Uhr**

Schuhegger, R., J. Colditz, I. Huber, C. Höller (Oberschleißheim) **11.50 Uhr**
Prävalenz von *Arcobacter* spp. in Bayern

Alter, T. (Berlin) **12.00 Uhr**
Ergebnisse zweier Sachverständigengespräche zur Entwicklung von Handlungsoptionen zur Reduktion von *Campylobacter* spp. im Geflügelbereich

Diskussion **12.10 Uhr**

Füllgrabe, R., H. Schönenbrücher, **12.20 Uhr**
A. Abdulmawjood, M. Bülte (Gießen)
Vergleichende Untersuchungen zu Dekontamination und kultureller Anzüchtung von *Mycobacterium avium* ssp.*paratuberculosis* (MAP) aus artifiziell kontaminierter Rohwurst

Saal „Werdenfels" - Lebensmittelhygiene

Bulander, K., A. Abdulmawjood, H. Schönenbrücher, M. Bülte (Gießen) — **12.35 Uhr**
Validierung und Optimierung von DNA-Extraktionsverfahren zum molekularbiologischen Nachweis von *Mycobacterium avium* ssp. *paratuberculosis* (MAP) aus Rinderkot

Diskussion — **12.50 Uhr**

Mittagspause — **13.00 Uhr**

Kabisch, J., R. Scheuer, W. Rödel (Kulmbach) — **14.00 Uhr**
Untersuchungen zur mikrobiologischen Wirksamkeit von Natriumnitrit bei Rohwursterzeugnissen

Knobloch, S., P.G. Braun (Leipzig) — **14.15 Uhr**
Einfluss der Hochdruckbehandlung auf mikrobielle Lipasen ausgewählter Verderbniserreger

Diskussion — **14.30 Uhr**

Messelhäußer, U., R. Zucker, D. Elmer-Englhard, W. Kleih, C. Höller, U. Busch (Oberschleißheim) — **14.40 Uhr**
Clostridium botulinum – Nachweisverfahren für den Einsatz in der Routinediagnostik

Schwarzmüller, C., B. Schalch, A. Stolle (München) — **14.55 Uhr**
Lactobacillaceae in einem Fleischwarenbetrieb – Ursache für den Verderb von Brühwürsten im Herstellungsprozess

Diskussion — **15.10 Uhr**

Kaffeepause — **15.20 Uhr**

Saal „Werdenfels" - Lebensmittelhygiene

Krause, N., M. Moravek, R. Dietrich, E. Märtlbauer (München) **15.40 Uhr**
Untersuchungen zur Produktion von *Bacillus cereus* Enterotoxinen bei verschiedenen Wachstumsbedingungen

Hammon, A., R. Pichner, M. Gareis (Kulmbach) **15.55 Uhr**
Vorkommen und Toxizität von *Bacillus cereus* in Gewürzen

Diskussion **16.10 Uhr**

Busch, U. (Oberschleißheim) **16.20 Uhr**
Einsatz von DNA-Chips in der amtlichen Überwachung

Böhmler, G., I. Thiem (Braunschweig) **16.35 Uhr**
Wirkungsbezogene Analytik in der Lebensmitteluntersuchung

Reichelt, H., K. Fehlhaber (Leipzig) **16.50 Uhr**
Anwendung von Schnelltestmethoden im Rahmen der Lebensmittelkontrolle

Trigo, D., I. Erol, R. Islam, **17.05 Uhr**
P. Josefowitz, G. Hildebrandt (Berlin, Ankara/TR)
Muskelfaserstruktur in Kochschinken-Erzeugnissen

Diskussion **17.20 Uhr**

Ende **17.40 Uhr**

Saal „Olympia" - Wildbrethygiene

Raßhofer, H. (OEJV, Frontenhausen) **8.30 Uhr**
Umsetzung der aktuellen EU-Vorschriften 852/53/2004 zur „kundigen Person" in der praktischen Jagdausübung

Baumer, G. (BJV und Veterinärdirektor, Amberg) **8.50 Uhr**
Umsetzung der aktuellen EU-Vorschriften 852/53/2004 zur „kundigen Person" aus der Sicht des Amtstierarztes

Stegmanns, T. (Stuttgart) **9.15 Uhr**
Wildbrethygiene bei Bewegungsjagden

Diskussion **9.30 Uhr**

Ellerbroek, L., A. Hensel, T. Herold, K. Nöckler (Berlin) **9.45 Uhr**
„Risiko Jagd?" – Eine bewertende Bestandsaufnahme

Paulsen, P., F.J.M. Smulders (Wien/A) **10.00 Uhr**
Die Expositionsabschätzung für Schrote in Fleisch von Feldhasen als Teil einer quantitativen Risikobewertung für physikalische Fremdkörper

Diskussion **10.15 Uhr**

Kaffeepause **10.25 Uhr**

Winkelmayer, R. (Bruck/A) **10.45 Uhr**
Wildfleisch-Direktvermarktung: Eine Leitlinie für die Gute Verfahrenspraxis nach den VO (EG) 852 und 853/2004

Vodnansky, M., F. Treml, **11.00 Uhr**
P. Paulsen, R. Winkelmayer (Brno/CZ, Wien/A, Bruck/A)
Zoonosen bei Feldhasen in Österreich, der Tschechischen Republik und in der Slowakei

Saal „Olympia" - Wildbrethygiene

Gneist, M. (Neustadt/A) **11.15 Uhr**
Die Tularämie in ausgewählten Regionen Niederösterreichs:
Beschreibung von drei Erkrankungsfällen

Deutz, A., T. Guggenberger (Murau/A) **11.30 Uhr**
Untersuchungen zur Verbreitung der Tularämie
unter dem Aspekt des Klimawandels

Diskussion **11.45 Uhr**

Wacheck, S., M. Fredriksson-Ahomaa, A. Stolle (München) **12.05 Uhr**
Wildfleisch – ein hochwertiges Produkt?

Türck, N., G. Klein, J. Apelt, V. Atanassova (Hannover) **12.20 Uhr**
Hygiene von frisch erlegtem und direktvermarktetem Wildfleisch

Bitter, J., G. Klein, J. Apelt, F. Reich, V. Atanassova (Hannover) **12.35 Uhr**
Bedeutung von sensorischen und mikrobiologischen Untersuchungen
bei der Beurteilung von Wildfleisch

Diskussion **12.50 Uhr**

Bucher, M., U. Messelhäußer (Oberschleißheim) **13.05 Uhr**
Amtliche Kontrolle von wildfleischverarbeitenden Betrieben, Teil I

Messelhäußer, U., M. Bucher, P. Kämpf, W. Kleih, G. Hauner, **13.15 Uhr**
C. Höller, H. Schreiner, H. Diepolder (Oberschleißheim, Erlangen)
Amtliche Kontrolle von wildfleischverarbeitenden Betrieben, Teil II:
Sensorische und mikrobiologische Beurteilung der Proben

Diskussion **13.25 Uhr**

Mittagessen **13.35 Uhr**

Saal „Olympia" - Rückstände & Resistenzen

Buettner-Peter, U. (Bonn) **14.00 Uhr**
Arzneimittelrechtliche Regelungen für lebensmittelliefernde Tiere

Große Beilage, T. (Essen) **14.15 Uhr**
Bestandsbetreuung lebensmittelliefernder Tiere

Bleyl, D. (Potsdam) **14.30 Uhr**
Problematik der oral zu verabreichenden Tierarzneimittel:
Anwendung und Stand der Wissenschaft und Technik

Kietzmann, M. (Hannover) **15.00 Uhr**
Rückstandsproblematik bei der Anwendung umgewidmeter Arzneimittel

Diskussion **15.15 Uhr**

Wallmann, J., U. Schröer, H. Kaspar (Berlin) **15.35 Uhr**
Nationales Antibiotikaresistenzmonitoring des Bundesamtes für Verbraucherschutz und Lebensmittelsicherheit – Optimierung der Lebensmittelsicherheit der vom Tier stammenden Produkte

Meyer, C., M. Fredriksson-Ahomaa, A. Stolle (München) **15.50 Uhr**
Antimikrobielle Resistenz bei *Yersinia enterocolitica*
Bioserotyp 4/O:3-Stämmen aus verschiedenen Quellen mittels Agardiffusionstest und Bouillon-Mikrodilutionsverfahren

Käsbohrer, A., B.-A. Tenhagen (Berlin) **16.05 Uhr**
MRSA bei Schweinen – Infektionsrisiko für den Verbraucher?

Diskussion **16.20 Uhr**

Saal „Olympia" - Rückstände & Resistenzen

Kuhn, K., B. Nowak, A. Seidel, **16.35 Uhr**
A. Lampen, G. Klein (Hannover, Großhansdorf, Berlin)
Nachweis Polycyclischer Aromatischer Kohlenwasserstoffe (PAKs)
in geräucherten Fleischwaren mittels chemischer Analyse und
in-vitro Testmethoden

Hamscher, G., S. Limsuwan, B. Prieß, **16.50 Uhr**
N. Tansakul, H. Nau, M. Kietzmann (Hannover)
Labor- und Feldstudien zur Untersuchung des Penetrationsverhaltens
von Phoxim- und Propoxurrückständen in Hühnereiern

Kaufmann, S., A. Stolle (München) **17.05 Uhr**
Schwermetallbelastung von Fleisch im Königreich Marokko:
Beispiel Kamelfleisch

Diskussion **17.15 Uhr**

Ende **17.30 Uhr**

Saal „Werdenfels" - Milchhygiene

Hamann, J., M. Schridde, F. Reinecke (Hannover) **9.00 Uhr**
Auswirkungen konventioneller und automatischer Melksysteme auf Milchsekretion und Milchleistung

Knappstein, K., K. Barth, E.-H. Ubben (Kiel, Westerau) **9.15 Uhr**
Beurteilung der Eutergesundheit von Milchziegen in Praxisbetrieben: Möglichkeiten und Grenzen

Walte, H.-G., G. Suhren (Kiel) **9.30 Uhr**
Bactoscan FC-Verfahren Ringtest: Auswirkung von robusten Verfahren auf die Präzisionsdaten

Diskussion **9.45 Uhr**

Grabowski, N.T., R. Redetzky, J. Hamann, G. Klein (Hannover) **10.00 Uhr**
Mastitis-Inzidenz erstkalbender HF-Kühe und Prävalenz euterpathogener Erreger

Krömker, V., J. Friedrich (Hannover) **10.15 Uhr**
Zur Ausscheidung von *S. aureus* über Milch aus infizierten Milchdrüsenvierteln

Redetzky, R., N.T. Grabowski, F. Reinecke, **10.30 Uhr**
J. Hamann, G. Klein (Hannover)
Zur Sekretion ausgewählter Milchinhaltsstoffe während der ersten Laktation eutergesunder Viertel von HF-Kühen

Diskussion **10.45 Uhr**

Kaffeepause **11.00 Uhr**

Saal „Werdenfels" - Milchhygiene

Loessner, M., K.-P. Kim, S. Günther (Zürich/CH) **11.20 Uhr**
Bakteriophagen ermöglichen effiziente Kontrolle von *Listeria monocytogenes*, *Salmonella enterica* und *Enterobacter sakazakii* in Lebensmitteln

Hammer, P., K. Knappstein, P. Teufel (Kiel) **11.35 Uhr**
Kontrolle von *Salmonella*-Kontaminationen im Umfeld einer Milchtrocknungsanlage

Diskussion **11.50 Uhr**

Kraemer, I.B., P. Kämpf, U. Messelhäußer, **12.00 Uhr**
W. Kleih, C. Höller, C. Bürk (Oberschleißheim, München)
Real-time-PCR-Nachweis von *Enterobacter sakazakii* in Speiseeis

Blüthgen, A. (Kiel) **12.15 Uhr**
Zum Carry-over von Aflatoxin B_1 in die Milch laktierender Schafe, Stuten und Ziegen vor dem Hintergrund des Grenzwertes für Aflatoxin M_1 in diätetischen Lebensmitteln

Diskussion **12.30 Uhr**

Mittagspause **12.40 Uhr**

Foyer 1.OG - Posterausstellung **14.00 Uhr**

Saal „Olympia" - Fischhygiene

Hoffmann, R. (München) **9.00 Uhr**
Tierärztliche Betreuung von Aquakulturen im Hinblick auf QM für Lebensmittel

Wedekind, H. (Starnberg) **9.20 Uhr**
Kulturtechnische Einflussmöglichkeiten auf die Fischqualität in der Aquakultur

Diskussion **9.40 Uhr**

Karl, H., U. Ruoff (Hamburg, Kiel) **9.50 Uhr**
Dioxine und dioxinähnliche PCB in Fischen und Fischerei-Erzeugnissen auf dem deutschen Markt – aktuelle Situation und Trends

Rehbein, H. (Hamburg) **10.05 Uhr**
Proteinanalytische Methoden zur Bestimmung der Erhitzungstemperatur von Fischerei-Erzeugnissen

Diepolder, H. (Erlangen) **10.20 Uhr**
Mikrobiologischer Status von Sushi

Diskussion **10.35 Uhr**

Kaffeepause **10.50 Uhr**

Saal „Olympia" - Fischhygiene

Gogus, U. (Ankara/TR) **11.10 Uhr**
Shellfish poisoning matters

Brenner, M., S. Ramdohr, M. Stede, S. Effkemann, **11.25 Uhr**
E. Bartelt, V. Etzel, A. Koehler (Bremerhaven, Cuxhaven)
Projekt *Mytifit* - Untersuchungen zur Zucht- und Verzehrsfähigkeit von Miesmuscheln (*Mytilus edulis*) aus Offshore Windparks

Özden, Ö., N. Erkan, M.C. Deval (Istanbul/TR) **11.40 Uhr**
Jahreszeitlicher Vergleich der Schwermetallakkumulation bei zwei Muschelspezies im Marmara-Meer, Türkei

Diskussion **11.55 Uhr**

Mittagspause **12.10 Uhr**

Foyer 1.OG - Posterausstellung **14.00 Uhr**

Saal „Werdenfels" - Fleischhygiene

Zweifel, C., R. Kohler, R. Stephan (Zürich/CH) **8.30 Uhr**
Prävalenz von *Campylobacter* spp., *Listeria* spp., *Salmonella* spp., Shigatoxin-bildenden *E. coli* (STEC) und Enteropathogenen *E. coli* (EPEC) bei Schlachtkaninchen

Grötzbach, B., M. Fredriksson-Ahomaa, A. Stolle (München) **8.45 Uhr**
Nachweis von pathogenen *Yersinia enterocolitica* in Rohfleisch und Rohfleischerzeugnissen von Rind, Schwein und Geflügel mit modernen molekularbiologischen Methoden

Hänsch, F., J. Hartung, B. Nowak (Hannover) **9.00 Uhr**
Betäubung von Puten mit Kohlendioxid in einem V-förmigen Tunnelsystem

Diskussion **9.15 Uhr**

Schalch, B., A. Stolle (München) **9.30 Uhr**
Dekontamination – Grundlagen und Entwicklung

Smulders, F.J.M. (Wien/A) **9.45 Uhr**
Dekontamination „revisited" als EU-Projekt

Stolle, A. (München) **10.00 Uhr**
Dekontamination von Fleisch verschiedener Tierarten im Oberflächensprühverfahren

Diskussion **10.15 Uhr**

Wichmann-Schauer, H., P. Hiller, A. Gervelmeyer (Berlin) **10.30 Uhr**
Lebensmittelbedingte Krankheitsausbrüche in 2006 – Informationen aus dem bundesweiten Erfassungssystem für Lebensmittel

Stein, M. (Zwickau) **10.45 Uhr**
Pilotprojekt: "Hygiene-Bonuspunktesystem der Stadt Zwickau Erfahrungen mit der bundesweit ersten öffentlichkeitswirksamen Ergebnisdarstellung der amtlichen Lebensmittelüberwachung"

Saal „Werdenfels" - Fleischhygiene

Knispel, B., J. Glatz, F. Grundhöfer, **11.00 Uhr**
S. Helble, A. Pastari, M. Rupp (Freiburg)
Erfahrungsbericht über ein landesweites Projekt mit dem Schwerpunkt HACCP und Rückverfolgbarkeit

Diskussion **11.15 Uhr**

Buettner-Peter, U. (Bonn) **11.30 Uhr**
Vorschlag für eine Verordnung über die Schaffung eines Gemeinschaftsverfahrens für die Festsetzung von Höchstmengen für Rückstände pharmakologisch wirksamer Stoffe in Nahrungsmitteln tierischen Ursprungs und die Aufhebung der EU-Verordnung 2377/90

Heberer, T. (Berlin) **11.45 Uhr**
Vorkommen und Verbleib von Human- und Veterinärarzneimittel-rückständen in der Umwelt und deren mögliche Relevanz für den gesundheitlichen Verbraucherschutz

Diskussion **12.00 Uhr**

Dietl, A., C. Höller, W. Kleih, A. Reiter-Nüssle, **12.10 Uhr**
B. Schalch, A. Stolle (München, Oberschleißheim)
Statuserhebung Schulverpflegung

Pelz, U., D. Trigo, G. Hildebrandt (Berlin) **12.25 Uhr**
Effektiver Nutzen neuer Medien in der Lehre am Institut für Lebensmittelhygiene der Freien Universität Berlin

von Breitenbuch, A., C.-P. Dickhaus, T. Blaha (Hannover) **12.40 Uhr**
Untersuchung über die Bedingungen für ein transparentes Fleischerzeugungssystem "from stable to table" unter Berücksichtigung von Tiergesundheit und Ernährungssicherheit

Diskussion **12.55 Uhr**

Abschlussdiskussion **13.05 Uhr**

Ende **13.15 Uhr**