the contrary, the "tragedy of the commons" applies to genetic resources. Many producers would like continued access to the genes of traditional breeds, but none has much incentive to bear the costs of collection and maintenance. The international community needs mechanisms to provide this global public good. Public-private partnerships hold promise; industry support for public initiatives will be essential.

Key Words: animal genetic resources, climate change, demand growth

543 Adaptation of the livestock sector to global climate change: Opportunities and options for animal genetic resources and management systems in developing countries. S. Fernandez-Rivera*, *Instituto Nacional de Investigaciones Forestales, Agrícolas y Pecuarias*, *Mexico City, D.F., Mexico*.

Model simulations are used to identify hot spots in developing countries where climate changes may result in new challenges to livestock production. Areas where climate changes may result in favorable situations for livestock production are also identified. The production systems practiced in those hot spots and their driving forces are described and their likely responses to temperature and rainfall variations are discussed. Variations in rainfall will have a major effect on quantity and quality of feed resources available, whereas higher temperatures will influence the prevalence of disease related factors and will have a direct effect on animals. Opportunities for livestock producers to adapt to those changes, with emphasis on animal genetic resources and specific traits are presented.

Key Words: climate change, livestock, developing countries

544 The role for animal genetic resources under global climate change conditions and rapid development of the livestock sector. I. Hoffmann*, *FAO*, *Rome, Italy*.

The livelihoods of one billion poor people are sustained by livestock. The livestock sector is the world's biggest land user and is associated with 18% of total greenhouse gas emissions. In large areas of developing countries, climate change threatens the livelihood of smallholders and pastoralists and may accelerate the erosion of animal genetic diversity. The paper will explore how animal genetic diversity is affected by climate change, and how it contributes to adapt to and mitigate climate change. Developed and developing countries differ in their adaptation capacity and the expected interactions between climate change adaptation and mitigation. Developing countries will have to apply a closer relationship between climate change adaptation and development policy. They also have weak capacity for high-tech breeding programes to increase their breeds' adaptation. Depending upon the ecosystem changes brought about by climate change and other pressures, the portfolio of breeds demanded by society will change. We assume that climate change itself, and the resulting disintegration of the components of (agricultural) ecosystems, together with human migration will increase the pressure to maintain wide access to animal genetic resources. Comprehensive policy frameworks that foster access to genetic resources as well as the development and use of appropriate technologies need to be developed. The recent adoption of the Global Plan of Action for Animal Genetic Resources provides for the first time an internationally agreed framework to promote creating these crucial conditions for the global livestock sector.

Key Words: animal genetic resources, climate change

545 The impact of global climate change, utilization of genetic resource management and livestock sector development on nutrition and health in developing countries. Y. Plante*[1] and H. Blackburn[2], [1]*Agriculture and Agri-Food Canada*, *Saskatoon, SK, Canada*, [2]*United States Department of Agriculture*, *Fort Collins, CO*.

Globally livestock employ 1.3 billion people and create livelihoods for one billion of the world's poor. In addition they provide one third of humanity's protein intake and therefore are a remedy for undernourishment. Livestock's growing role in developing country food security is evidenced by an increase (from 1960 to 2003) of milk (70%) and meat (190%) consumption when compared to an 18% increase in cereal consumption during the same time period. The full ramifications of global climate change, its interaction with animal genetic resources for food and agriculture and the ultimate ability of local production systems to provide animal products and to sustain food security or income to developing country smallholder farmers and landless people is unclear. Predicted climate changes will affect soil erosion and fertility, crop, forage and livestock management in terms of decision making regarding water resources, seeding and harvesting different varieties, pest and disease control, and locally adapted livestock species and breeds, especially in tropical and sub-tropical regions. Clearly mitigating actions are needed to buffer such events and to insure that livestock's contribution to health, nutrition and economic growth continues. It has been suggested that livestock producers will make the necessary adjustments, for example by abandoning traditional cattle breeding and adopting small ruminant husbandry practices. However, a number of policy and technological changes will have to occur for a systematic transition to new animal production systems so that livestock smallholders and pastoralists may be protected against greater levels of food insecurity. How these producers can buffer themselves from such changes is important and exploration into this subject is crucially needed so that governments and multilateral agencies can work toward potential solutions.

Key Words: climate change, animal genetic resources, food security

Lactation Biology: Lactation Biology 2

546 Prolactin, insulin and cortisone regulate expression of GLUT8 gene in bovine mammary explants. K. Zhao*, H. Y. Liu, and J. X. Liu, *Institute of Dairy Science, Ministry of Education Key Laboratory of Molecular Animal Nutrition, Zhejiang University*, *Hangzhou, P.R. China*.

Lactogenic hormones are known to regulate milk synthesis and secretion in lactating dairy cows, but knowledge about their possible effects on glucose transporters in mammary gland at tissue level remains controversial. GLUT8 is a newly identified member of the facilitative glucose transporter family, and may play an important role in glucose uptake in the lactating mammary gland. This study was aimed at investigating effects of the three primary lactogenic hormones, prolactin, insulin, and cortisone, on expression of GLUT8 mRNA in cultured bovine mammary tissue taken from the mid-lactating dairy cows. The mRNA expression was determined by SYBR green method of quantitative reverse tran-

scription PCR (ABI 7500). The explants were cultured in DMEM/F12 medium containing 10% fetal bovine serum (FBS) and treated by prolactin (0, 50, 1000, 5000 ng/ml), insulin (0, 5, 50, 500, 5000 ng/ml), and cortisone (0, 50, 1000, 10000 ng/ml), respectively for 48 hr after 24 hr starvation of FBS. Compared with control (no hormone), prolactin (50, 1000 ng/ml) decreased GLUT8 gene expression ($P<0.05$), but had no effects at 5000ng/ml. Insulin above 50ng/ml doubled the abundance of GLUT8 mRNA, while 5ng/ml had no effect. The expression of GLUT8 mRNA was enhanced firstly and then depressed with the increasing level of cortisone, with highest abundance at 1000ng/ml of cortisone ($P<0.05$). The regulation pattern and sensitivity of GLUT8 to lactogenic hormones were different from those of GLUT1 that is the predominant glucose transporter in bovine mammary gland (data not shown). The results demonstrate that GLUT8 expression may be regulated by lactogenic hormones in a dose-dependent manner, suggesting that lactogenic hormones may influence glucose transportation in bovine mammary gland through regulation of transporter gene expression.

Key Words: bovine mammary gland, GLUT8 gene expression, lactogenic hormone

547 Effect of the milking-induced prolactin release on galactopoiesis in dairy cows. V. Lollivier*[1], R. M. Bruckmaier[2], P. Lacasse[3], and M. Boutinaud[1], [1]*INRA, AGROCAMPUS OUEST, UMR1080, St. Gilles, France*, [2]*University of Bern, Bern, Switzerland*, [3]*AAFC, Dairy and Swine R&D Centre, Sherbrooke, Canada*.

In ruminants, milking induces the release of prolactin (PRL). Milking-induced PRL release decreases as lactation advances and PRL is a survival factor for mammary epithelial cells (MEC) of cows, suggesting a galactopoietic role of this hormone. Nevertheless, suppression of PRL by bromocriptine has produced ambiguous effects on milk yield in cows. To assess the effect of inhibition of PRL release in lactating dairy cows, nine Holstein cows were assigned randomly to treatments during 3 5-d periods: 1)daily i.m. injection of 2 mg of Quinagolide (a PRL release inhibitor), 2)daily i.m. injection of 2 mg of Quinagolide and twice a day (milking time) i.v injection of PRL (2µg/kg of body weight),3) daily injection of vehicle as control. Blood and milk samples were harvested at milking. MEC were purified from milk. Daily injections of Quinagolide reduced milking-induced PRL release ($P<0.05$). The amount (area under curve) of PRL during milking after the PRL injections was similar to that of endogenous PRL discharges in control treatment. Quinagolide decreased milk production ($P<0.05$), milk protein content ($P<0.05$) and milk lactose content ($P<0.05$), without modification of milk fat composition or fat globule size. PRL injections had no effect on milk yield and milk fat composition but tended to increased milk protein content ($P<0.10$). Injections of Quinagolide increased the number of MEC harvested from milk ($P<0.05$) but PRL injections tended to decrease it ($P=0.10$). PRL injections also increased viability of MEC harvested from milk ($P<0.05$). Injections of Quinagolide decreased kappa-casein ($P<0.05$) and alpha-lactalbumin ($P<0.05$) mRNAs in milk MEC. Exogenous PRL have no affected the level of these mRNA. In conclusion, chronic administration of Quinagolide reduces milk production in dairy cows by affecting MEC activity. PRL injections at milking time were not sufficient to restore milk yield but influenced the viability of MEC purified from milk.

Key Words: prolactin, milking, dairy cows

548 Effects of unilateral frequent milking of dairy heifers during early lactation. J. B. Wright*, E. H. Wall, and T. B. McFadden, *University of Vermont, Burlington*.

In multiparous cows increasing milking frequency from 2X to 4X/d during days 1 to 21 of lactation stimulates an immediate response in milk yield that partially persists throughout lactation. However, it is unknown if dairy heifers respond similarly. The objective of this study was to investigate the response of dairy heifers to frequent milking during early lactation using a half-udder design. Holstein heifers were assigned at parturition to unilateral frequent milking (UFM). On days 1 to 21 of lactation Heifers were milked twice daily at 0130 h and 1330 h, with additional milking of the right udder half at 0430 h and 1630 h. Thereafter, heifers were milked twice daily. Half udder milk yields and teat end scores were measured weekly on days 1 to 35 of lactation. Teat ends were scored on a scale of 0 to 4, with 0 representing no apparent teat end damage and 4 representing severe damage to the teat end. A 1-sided paired t-test was used to compare milk yields of 2× vs. 4× udder halves. Effects of time, milking frequency and their interaction on teat end scores were analyzed using the mixed procedure of SAS. During UFM milk production of 4× udder halves rapidly increased relative to that of 2× udder halves ($P < 0.001$). After cessation of UFM the difference in milk yield was no longer significant at day 28, but was partially restored by day 35. Teat end scores increased over time ($P < 0.001$), but were not effected by milking frequency. We conclude that frequent milking of heifers during early lactation stimulates an increase in milk yield that partially persists after treatment. These results are similar to those of mature cows.

Table 1. Effect of Unilateral Frequent Milking on Milk Yield and Teat End Score in Heifers

DIM	1	7	14	21	28	35
n =	5	7	7	7	6	6
4X-2X, kg/d	-0.2	2.0*	4.1*	3.3*	0.5	0.9^{+}
Teat Score	0.3	0.6	1.0	1.1	1.6	1.6

*$P < 0.01$, $^{+}P < 0.1$

Key Words: frequent milking, heifer, teat end

549 Effects of reduced frequency of milk removal on gene expression in the bovine mammary gland. M. Littlejohn*[1], C. Walker[1], H. Ward[2], K. Lehnert[2], R. Snell[2], G. Verkerk[1], R. Spelman[3], D. Clark[1], and S. Davis[2,3], [1]*DairyNZ Ltd, Hamilton, New Zealand*, [2]*ViaLactia Biosciences Ltd, Auckland, New Zealand*, [3]*Livestock Improvement Corporation, Hamilton, New Zealand*.

In mammals, reduced suckling frequency by the offspring initiates the down-regulation of milk synthesis and the induction of apoptotic pathways and structural remodeling of mammary tissue. This effect on milk synthesis and secretion has been shown to be mediated through local (intra-mammary) mechanisms, however the physiological triggers and discrete signaling pathways that comprise the response to milk removal frequency remain largely undefined. To gain insight into the pathways and individual molecules involved in this response, an analysis of mammary gene expression was conducted in 12 lactating cows milked at two different frequencies. Animals milked twice a day were sampled by mammary tissue biopsy and then milked once daily for five days. A second biopsy was then taken from the adjacent rear-udder quarter of these animals, allowing changes in gene expression to be assessed within each animal. Expression analysis was conducted

using Agilent bovine oligonucleotide arrays representing 21,495 transcripts, and revealed a range of genes differentially expressed as a result of less frequent milk removal. These changes included an increase in abundance of transcripts related to apoptotic signaling (NFKB, JUN, ATF3, GADD45A, IGFBP5, TNFSF12A) and mechanical stress and epithelial tight junction synthesis (CYR61, CTGF, CLDN4, CLDN8). Concomitant with a reduction in milk yield, a downregulation of molecules related to milk synthesis (LALBA, B4GALT1, UGP2, CSN2, GPAM, LPL) was also observed. Quantitative real time PCR was used to assess the expression of 13 genes in the study, and all were highly correlated ($p<0.05$) with values derived from array analysis. It can be concluded that the apoptotic signaling pathways characteristic of recognizable involution and mammary regression occur early in response to a reduction in milk removal frequency. Further, these results suggest that mechano-signal transduction cascades (initiated as a result of udder distension) play roles in initiating the apoptotic signaling networks that orchestrate this response.

Key Words: milking frequency, involution, gene expression

550 The ability of exogenous growth hormone to maintain milk production during prolonged lactation in the mouse is more evident with reduced nursing frequency. D. L. Hadsell*[1], W. Olea[1], A. F. Parlow[2], and R. J. Collier[3], [1]*Baylor College of Medicine*, *Houston, TX*, [2]*Harbor-UCLA Medical Center*, *Torrance, CA*, [3]*The University of Arizona*, *Tucson*.

Although growth hormone (GH) increases milk production in dairy animals, the milk production response of lactating rodents to this treatment has been variable. Milk removal frequency in the lactating mouse is about 10-fold higher than that of lactating dairy cows. The hypothesis tested in this study was that the ability of GH to stimulate milk production during prolonged lactation would be greater in mouse dams subjected to reduced nursing frequency than in dams allow to nurse ad-libitum. The growth of 8-day-old crossfoster litters maintained on groups of lactating mice was studied either from day 14 to 21 postpartum, or from day 21 to 27 postpartum using a litter cross-fostering protocol that prevents natural mammary involution. There were four treatments that consisted of ad-libitum (AL) or reduced nursing frequency (4×) in combination with subcutaneous injections of either saline (SAL) or recombinant murine GH (GH). The GH was tested at 2 doses of either 6 or 18 mg/kg/day. Reduced Nursing frequency caused a dramatic decrease in litter gain ($P<0.0001$) in both SAL- and GH-treated dams. At 6 mg/kg/day, GH failed to significantly increase day 14 to 21 litter gain in either AL or 4× dams (12.4±1.4, 15.4±1.2, 24.5±1.6 and 25.5±0.9 g for 4×-sal, 4×-GH, AL-SAL, and AL-GH, respectively). At a dose of 18 mg/kg/day, GH increased day 14 to 21 litter gain in 4× ($P<0.02$), but not in AL litters (10.56±0.8, 16.03±1.4, 30.1±1.9, and 30.1±0.8 g for 4×-SAL, 4×-GH, AL-SAL, and AL-GH, respectively). Litter gain from day 21 to 27 was increased by GH in both AL ($P<0.01$) and 4× ($P<0.001$) litters (2.4±1.9, 11.0±0.7, 13.5±0.5, and 18.8 g for 4×-SAL, 4×-GH, AL-SAL, and AL-GH, respectively). These results support the conclusion that the ability of GH to stimulate milk production in lactating mice is affected both by stage of lactation and the frequency of milk removal. *This project was supported by National Research Initiative Competitive Grant no. 2007-35206-17831 from the USDA Cooperative State Research, Education, and Extension Service.*

Key Words: milking frequency, growth hormone, mouse

551 Mammary transcript profiles due to prepartum dietary energy level and bacterial lipopolysacharide challenge in dairy cows early postpartum. D. E. Graugnard*, S. L. Rodriguez-Zas, R. E. Everts, H. A. Lewin, J. K. Drackley, and J. J. Loor, *University of Illinois*, *Urbana*.

Immunosuppression renders cows highly susceptible to mastitis pathogens as well as metabolic diseases after parturition. We hypothesized that plane of dietary energy prepartum can affect tissue response to inflammatory challenges through changes in gene expression. Twenty–eight Holstein cows with average composite SCC of ~128,000 in the previous lactation were assigned (n = 14/diet) to a control (high-straw; NE_L = 1.52 Mcal/kg) or moderate-energy (ME; NE_L = 1.64 Mcal/kg) diet during the entire dry period. All cows were fed a common lactation diet (NE_L = 1.69 Mcal/kg) postpartum. At 7 DIM, cows (n = 7/prepartum diet) were assigned to receive an intramammary bacterial lipopolysacharide (LPS) challenge (200 μg) in one rear mammary quarter or served as controls. Cows used were bacteriologically–negative in all mammary quarters. A percutaneous mammary biopsy was collected at 2 h post–LPS for transcript profiling using a 13,257 annotated bovine oligonucleotide microarray. LPS challenge of cows fed ME prepartum resulted in >100 differentially expressed genes (DEG, $P < 0.01$). Among DEG, the most enriched biological functions were response to stimulus (n = 21) and immune response (n = 11). We also observed genes with higher mRNA abundance due to LPS that were related with regulation of immune response (e.g. *MBP, SIT1*), inflammatory response (e.g. *TLR9*), and chemotaxis (e.g. *CXCL2*). In the comparison of ME vs. control–fed cows receiving LPS postpartum there were >20 DEG due to prepartum diet. The most affected biological function among DEG was transport regulation (n = 10). Genes with higher mRNA due to feeding the control diet prepartum were involved primarily in immune response regulation (e.g. *CD59*). Results showed that a mammary inflammatory challenge early postpartum caused rapid alterations in tissue gene expression profiles. However, the transcriptomic response to inflammation was not greatly affected by prepartum dietary energy level.

Key Words: transcriptomics, inflammation, mastitis

552 Fluoxetine and phenelzine disrupt tight junctions in primary bovine mammary epithelial cells. L. L. Hernandez*[1], R. J. Collier[2], and N. D. Horseman[1], [1]*University of Cincinnati*, *Cincinnati, OH*, [2]*University of Arizona*, *Tucson*.

Serotonin (5-HT) acts via autocrine-paracrine mechanisms on mammary epithelial cells in a variety of species. In human mammary cells, 5-HT treatment disrupts tight junctions (TJ) in a biphasic manner. Low doses and short exposures increase TJ integrity, and high, sustained treatment disrupts TJ. TJ remain "tight" throughout lactation, preventing leakage of milk components out of the luminal space, and become "leaky" during involution. The 5-HT reuptake transporter and the enzyme monoamine oxidase (MAO) regulate 5-HT concentrations in the extracellular space by allowing 5-HT to re-enter the epithelium to be degraded to 5-hydroxyindole acetic acid. In these experiments, we investigated the effects of treatments with fluoxetine, a selective 5HT reuptake inhibitor (SSRI), and phenelzine, a monoamine oxidase inhibitor (MAOI) on transepithelial resistance (TEER) in primary bovine mammary epithelial cell (BMEC) cultures. BMEC were grown on Transwells® for 6 d, and then treated with 40 and 400 μM, and 1.4 mM SSRI, 40 and 400 μM, and 1.4 mM MAOI, and 40 and 400 μM, and 1.4 mM SSRI in combination with MAOI for 48 h. Media was exchanged daily and TEER was measured prior to media changes. Percent change was calculated for all treatments relative to the control. The 40 μM SSRI treatment resulted in 15% and 27% decreases in TEER, 24 and 48 h

after treatment, respectively ($P < 0.001$). The 400 μM and 1.4 mM SSRI treatments resulted in 86% decreases in TEER 24 h after treatment, and decreased 75% after 48 hr after treatment ($P < 0.001$). The 40 μM, 400 μM, and 1.4 mM MAOI treatments decreased TEER 1.63, 17.8, and 12.2% respectively, 24 h after treatment and 16.2, 37.9, and 41.9% respectively, 48 hr after treatments. Treatment of pBMEC with SSRI + MAOI, resulted in similar decreases in TEER seen with SSRI treatment alone. In conclusion, while both SSRI and MAOI resulted in tight junction disruption in pBMEC. SSRI resulted in larger decreases in TEER relative to the control when compared to MAOI treatments. *Project supported by NRI Grant #2007-35206-17898 from USDA-CSREES.*

Key Words: serotonin, tight junctions, serotonin reuptake transporter

553 Detection of bioluminescent *Staphylococcus aureus* through bovine mammary gland tissue *ex vivo*. J. Curbelo*, K. Moulton, E. Schenck, and S. Willard, *Mississippi State University*, *Mississippi State*.

Mastitis infections caused by *Staphylococcus aureus* (*S. aureus*) causes serious problems for the dairy industry due to its high degree of pathogenicity, and new models are needed for monitoring mastitis infections. The objectives of this study were 1) to conduct photonic imaging (PE) of a transformed bioluminescent *S. aureus*-lux (Caliper Life Sci.) through bovine mammary gland tissue (BMGT); and 2) to evaluate the significance of an optical clearing agent (OCA; corn syrup, 75%) in increasing transference of PE through BMGT. To accomplish this, n=10 bovine mammary quarters with intact teats were excised in half (n=20 sections) and placed over 5 ml of concentrated *S. aureus*-lux inoculums (3.5×10^{14} CFU) inside a black plastic box (opened top) for imaging and PE quantified in relative light units per second (RLU/s). Then, one of the teat sections was treated with OCA for 3 h and the other section was used as control and imaged, as previously described. Due to the high degree of variation of PE between the 20 teat sections, they were categorized by color (black, mixed and white). The mean PE of all teats (non-OCA treated) was 2.30 ± 0.53 RLU/s. However, when analyzed by color, PE through black teats were significantly lower than mixed and white teats ($P=0.01$; 0.68 ± 0.08 vs. 3.77 ± 1.50 and 3.48 ± 0.78 RLU/s, respectively). In addition, when applying OCA to teats, resulted in a greater detection of PE numerically, but not statistically ($P=0.18$); however when the significance of the OCA was evaluated by color, a higher PE was found in mixed colored teats treated with OCA vs non-OCA treated ($P=0.01$; 1.44 ± 0.12 vs 0.53 ± 0.05 RLU/s, respectively). In summary, this study indicates that detection of *S. aureus*-lux through BMGT is questionable due to the low PE detected; however, higher PE were detected through white and mixed colored teats. The OCA used in this study did not increase PE overall. For future studies, the color of the teats may need to be considered due to differences in PE transference, and further studies are needed to enhance penetration of the dense tissue of the mammary gland for photonic pathogen detection.

Key Words: *S. aureus*, biophotonics, mammary gland tissue

Nonruminant Nutrition: Minerals and Vitamins

554 Effects of phytase supplementation on apparent and standardized total tract digestibility of P in corn, soybean meal, and distillers dried grains with solubles (DDGS) fed to growing pigs. F. N. Almeida* and H. H. Stein, *University of Illinois*, *Urbana*.

An experiment was conducted to measure the apparent total tract digestibility (ATTD) and the standardized total tract digestibility (STTD) by growing pigs of P in corn, soybean meal (SBM), and distillers dried grains with solubles (DDGS) without and with supplementation of microbial phytase. Seven diets were prepared. Two diets were based on corn, 2 diets were based on SBM, 2 diets were based on DDGS, and 1 diet was a P-free diet. Corn, SBM, or DDGS were the only sources of P in the diets. One of the diets with each ingredient contained no microbial phytase while the other diet contained 500 units/kg of phytase (Optiphos, Enzyvia LLC, Sheridan, IN). A total of 42 growing barrows (initial BW: 13.5 ± 3.9 kg) were randomly allotted to the 7 dietary treatments with 6 pigs per treatment. All pigs were fed experimental diets for 10 d with the initial 5 d being an adaptation period to the diet, whereas feces were collected quantitatively during the final 5 d of the experiment using the marker to marker procedure. The ATTD of P was calculated for corn, SBM, and DDGS and the effect of microbial phytase was calculated as well. The basal endogenous losses of P (ELP) were measured from pigs fed the P-free diet and values for ATTD of P in the 6 P-containing diets were corrected for the basal ELP to calculate STTD values for P in each of these diets. Results showed that the addition of phytase increased ($P < 0.001$) ATTD of P from 19.9 to 57.8% in corn and from 41.5 to 68.4% in SBM, but the ATTD of P in DDGS without phytase (68.6%) was not different from the ATTD of P in DDGS with phytase (71.0%). The ELP was 199 mg/kg DMI. The addition of phytase also increased ($P < 0.001$) STTD of P in corn and SBM (from 26.4 to 64.4% and from 48.3 to 74.9%, respectively), but the STTD values of P in DDGS without and with phytase (72.9 and 75.5%, respectively) were not different. In conclusion, the addition of phytase increased the ATTD and STTD of P in corn and SBM, but had no influence on the ATTD and STTD of P in DDGS.

Key Words: digestibility, pigs, phosphorus

555 Determination of the stability of Zn, Mn, Cu and Fe glycinates in aqueous solution by electrospray QqTOF mass spectrometry. S. Oguey*[1], V. Vacchina[2], R. Lobinski[3], and D. Bravo[1], [1]*Pancosma*, *Geneva, Switzerland*, [2]*UT2A*, *Pau, France*, [3]*CNRS*, *Pau, France*.

The crystalline structure of trace mineral complexes based on glycine (BT) was well known, however setting analysis methods of the products in diluted media (feed, stomach juice, plasma,...) required a previous identification and characterisation of the complexes existing in aqueous solution. The chemical structure of BT with elements of the first transition series (Fe, Cu, Mn and Zn) in aqueous solution was investigated by mass spectroscopy with an electrospray QqTOF mass spectrometer (ESI-QqTOF-MS) either in full scan TOF mode or in tandem mass spectrometry (MS/MS) mode. Each BT was dissolved in a 50% methanol and 10 mM ammonium acetate buffer at pH 7. BT Fe solutions were previously degassed to prevent iron oxidation and iron hydroxide precipitation. Once the chemical structures determined at pH 7, the pH of the BT solutions was varied from 2 to 7 by step of 1 in order to determine the stability of the complexes in solution, and the evolution of the amount of the complex and free glycine ions was observed. BT in aqueous solution at pH 2 were still detected as glycine complexes. The chemical structure was slightly different from the structure found in solid, except for manganese which was identical. Their general formula