Nutritional Profiles: A New Approach For Dairy Herds[a]

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Veterinarians, producers and nutrition consultants alike seem to be interested in extracting pertinent information relative to herd nutrition and health status from simple blood tests. The Compton Metabolic Profile (CMP) has traditionally been used in this approach.[1] The original intent of the CMP was to: (1) monitor metabolic health of the herd, (2) help diagnose metabolic problems and production diseases, and (3) identify metabolically superior cows.[1,2] A "metabolic profile" is defined as a series of specific analytical tests run in combination and used as a diagnostic aid. Use of a metabolic profile is the result of technologic improvements in analytical instrumentation which can complete multiple analyses in a short time period. Selection criteria for biochemical parameters to be run within a profile include: stability of the metabolite; accuracy and ease of analytical procedure; low cost and consistent relationship between metabolite and nutrient status or some metabolic pathway involved in producing signs associated with metabolic disease.[3,4] In addition to these criteria, factors such as age, sex, genotype, diurnal and prandial variation and environmental stress should have minimal effect on metabolite concentrations.

Metabolic profiles are commonly used in all veterinary hospitals as an aid in diagnosis of individual animal health problems. Application of this diagnostic procedure on a herd basis has been questioned relative to its validity and sensitivity in defining a problem as well as its cost. Unfortunately in many herd situations, blood analyses are used preferentially in lieu of other more appropriate diagnostic procedures such as ration evaluation and physical exams and without regard for proper technique to ensure sound diagnostic information. However, blood metabolite analysis can reveal some useful information if properly interpreted in conjunction with animal and ration evaluations. This presentation will review the application and limitations of the metabolic profile and describe an alternative approach to using this procedure.

Compton Metabolic Profile

The CMP involved collecting 7-to-10 blood samples from 3 predefined groups of dairy animals, i.e., dry, peak lactation and midlactation, and having selected metabolites (Table 1) measured.[1] From the test results, means for each metabolite are calculated for each respective group and compared to reference values. Reference values for metabolites are determined statistically from means across all herds and production stage data with the range including ± 2 standard deviations around the mean.[5,6] Seven animals are considered the minimum number sampled to be statistically significant for interpretation. As one might expect, completing 11-to-13 biochemical tests on 21 individual samples is extremely expensive ($200 to >$400), even with automated equipment. Blowey proposed a smaller scale version of the CMP scheme to address cost concerns and maintain some diagnostic capability.[7,8]

Table 1.

Compton Metabolic Profile:

Packed Cell Volume
Hemoglobin
Glucose
Blood Urea Nitrogen
Total Protein
Albumin
Calcium
Inorganic Phosphorus
Magnesium
Potassium
Sodium
Copper
Iron

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This test procedure, besides being expensive, had some perceived limitations as a diagnostic procedure. Three major sources of variation in blood metabolite concentration were found to be herd of origin, level of milk production (stage of lactation) and season of year.[4,5,9] These factors needed to be addressed in order to properly interpret the data. Other documented sources of variation in blood metabolite concentrations include: parity, time relative to calving, pregnancy status, environmental conditions, dietary nutrient interrelationships and sample handling.[3,6,10] If one is to obtain useful information from a metabolic profile, all of these parameters will need to be taken into account. This suggests that consideration must be given to appropriate animal selection and careful interpretation of the data. The originators of this procedure suggested that both a nutritionist and veterinarian be involved.

The CMP has generally received positive endorsements as a diagnostic aid from studies outside the United States.[1,11-13] In contrast, results of metabolic profiles in studies completed in the US have generally been less than enthusiastic about their potential diagnostic value.[10,14,15] Reasons for this difference in perception are varied and include feeding and management differences between European and US dairies (e.g., pasture-based vs. confinement feeding), study design and study objectives. For the most part, studies completed in the US have focused on the use of metabolic profiles as determinants of nutritional status. Suggested uses of the metabolic profile include: (1) to predict the possibility or presence of metabolic disease; (2) to assess fertility status and (3) to assess nutritional status.[3] These are somewhat different from the intended use of the CMP as defined by the original authors.

Metabolic profiles have not proven to be good predictors of nutritional or fertility status, which may explain why this test has not been favorably accepted in the US. In fact, the developers have suggested that the metabolic profile test is not a nutritional test.[6] Other studies also caution against making strong inference between blood metabolite concentrations and dietary adequacy. Although studies have shown a limited number of significant associations between dietary and blood nutrient status.[3,10,16] Many specific nutrients can influence some aspect of fertility.[17-20] However, few studies can show consistent associations between metabolite status and fertility. This is most likely a result of the myriad of other factors and interrelationships which influence reproductive performance.

A problem with analysis of the metabolic profile is the desire for simplicity in interpretation. Unfortunately, the determination of normal and abnormal is not as black and white as we would desire. Abnormal metabolite values are defined as those outside of the predefined normal reference range. However, there are discrepancies among studies as to what should be defined as the "normal range". The mean ± 2 standard deviations (95%) has been the standard definition; although some studies have advocated ± 1.3 or 1 standard deviation depending upon the metabolite of concern.[3] Wide ranges result in a failure to identify abnormal herds (false negative) whereas more narrow ranges may result in calling normal herds abnormal (false positive). A lack of sensitivity and specificity in differentiating normal from abnormal herds has been the primary detractor to accepting this procedure as a useful diagnostic aid. Data reported by Payne show that herds with no problems are not well differentiated in contrast to problem herds (Table 2).[5] This supports the contention that metabolic profiling data need to be coordinated with herd history and animal evaluations.

Table 2. Metabolic profile results on herds with or without metabolic disease.[1]

Herd Problem	Total Herds	Normal Profile	Abnormal Profiles[2]
None	30 (100%)	13 (43.3%)	17 (56.7%)
Parturient Paresis	10 (100%)	0 (0%)	10 (100%)
Infertility	20 (100%)	2 (10%)	18 (90%)
Ketosis	7 (100%)	0 (0%)	7 (100%)

[1]Data adapted from Payne[1]
[2]Abnormal represents one or more mean values for any group deviating by more than 2 standard deviations from the reference mean.

METABOLIC PROFILES - Procedures

Animal Selection

The goal of any metabolite profiling is to obtain the "population" mean and determine dynamic changes over differing physiologic states. To obtain this we need to sample large numbers of animals. Initially, cost is the main deterrent to large animal numbers; however, why not pool samples since we are interested in the mean value and not individuals? Samples can be pooled by appropriate physiologic states to allow interpretation of dynamic changes in "population" means over a period of time. For example to address a fresh cow problem, pooled samples are collected from recently dry cows (>7 days following dry-off), close-up dry cows (<3 weeks and >5 days prior to calving) and fresh cows (< 30 or < 60 days in milk). Clinically affected cows can be sampled and pooled and compared to normal cows that are matched according to parity and stage of lactation. Other appropriate sample pools can be determined given the specific problem to be addressed. By pooling samples you are obtaining information from a greater number of animals for much less cost. Rather than the standard 21 samples to calculate 3 group means, you may submit 3 pooled samples which represents a mean of 10 to 20 animals each. The only negative part to this method is the loss of statistical evaluation, i.e., population vari-

ance. However, this is not a major limitation. Proper identification of the appropriate animal groups or pools is absolutely critical if one is to obtain useful data.

For data from these pooled samples to be relevant, all cows should be equally represented. The author prefers to have samples drawn only from visually nonclinical animals to more accurately represent the population. If needed, you could pool both clinical and nonclinical animals within a given group for comparison. To be able to appropriately interpret changes from one physiologic state to another at a single point in time, all animals should have been exposed to the same diets and management environments. This means to say that the fresh cows sampled today received the "same" diet that the early dry cows are currently receiving. If a dietary change was made recently, then comparisons between physiologic states is not appropriate. If no changes were made, then compare dynamic changes in the "population" means for specific metabolites in accordance with clinical signs and ration evaluation.

Sample Collection

Sample collection and handling are important if one is to expect useful diagnostic information in return. Blood samples should be taken from either the jugular or coccygeal veins with a minimal amount of stress. Blood samples from the mammary veins are not appropriate given the loss of nutrients into the mammary gland. Lower concentrations of phosphorus and potassium have been documented in jugular compared to coccygeal blood samples as a result of salivary gland uptake.[4,6,21] Time of sampling relative to feeding and feeding management may also influence metabolite concentrations and should be considered in the decision process of when to sample. If herds are being repeatedly sampled as a monitoring tool, samples should be taken at approximately the same time of day to minimize diurnal and prandial variation.

Vacuum tubes are the most common and easiest form of sample collection. A variety of vacuum tubes are available. Vacuum tubes are color coded for specific diagnostic test procedures based on the specific anticoagulant or additive present in the tube (Table 3). Most commonly a single serum or plasma sample is collected; however, in some cases a whole blood sample may be desired. Extreme efforts should be taken to prevent hemolysis of the sample. All samples should be properly identified with animal and group identification and date of collection. Other pertinent information for interpretation of the metabolic profile would include: animal age, lactation number, milk production level, milk composition, days in milk, pregnancy status and body condition score.

Table 3. Description of blood collection tubes used for metabolic profiles.

Stopper Color	Additive	Sample Obtained	Intended Use/Disadvantages
Red	None	Serum	Routine use for all tests./ Prolonged clot exposure results in ↓ glucose, Ca and ↑ phosphorus. Hemolysis problems in poorly handled samples.
Gray	Na Fluoride or K Oxalate	Serum	Glycolytic Inhibitor for sensitive glucose analysis
Royal Blue	plastic stopper ± Na Heparin	Serum or Plasma	Trace mineral analysis
Lavender	EDTA	Whole Blood Plasma	Routine use for Complete Blood Count/ EDTA chelates Ca, Mg and ↓ enzyme activities
Green	Na Heparin	Plasma Whole Blood	Routine analyses for either plasma or whole blood / No effect on metabolites

Analytical Tests

As for the metabolite assays, the author routinely runs a complete profile as defined by the Clinical Pathology Laboratory, College of Veterinary Medicine at Oregon State University which includes: blood urea nitrogen (BUN), creatinine, glucose, total protein, albumin, total bilirubin, alkaline phosphatase (ALP), creatine kinase (Ck), (-glutamyltransferase (GGT), aspartate aminotransferase (AST), sodium (Na), potassium (K), chloride (Cl), calcium (Ca), inorganic phosphorus (P) and magnesium (Mg). In addition, total cholesterol and nonesterified fatty acids (NEFA) are determined. All these tests only require serum although they can also be run on plasma from a green stoppered tube. The author is looking specifically for changes in energy balance (NEFA values and BCS changes), protein status (BUN, Creatinine, Total protein, Albumin, Ck), macromineral status (K, Ca, P, Mg) and liver function (cholesterol, GGT, AST, Bilirubin).

INTERPRETATION OF METABOLIC PROFILES

Reference values

Interpretation of metabolic profiles requires some standard reference values for comparison. These reference values should represent the population mean and variation from a defined population of animals clinically evaluated to be free of disease and other health problems. The reference range represents the population mean ± 2 standard deviations. Each population mean needs to be statistically analyzed for a normal population distribution. Median values should be used in place of means for metabolites not showing a normal distribution.[16] A question needs to be posed at this point: Since it has been shown that stage of lactation has significant effects on blood metabolite concentrations, should refer-

ence values be established accordingly? At present this has not been done. Research is currently underway to ascertain if metabolite concentrations are significantly different from present established reference values.

Pooled mean metabolite values are compared to the reference values to assess differences. At this point, there is need for some intuitive interpretation. For example, if the Ca concentration for fresh cows is 8.7 mg/dL and the reference range is 9 to 12 mg/dL, this might be interpreted to suggest a potential problem with subclinical hypocalcemia. The measured mean of 8.7 mg/dL represents a population with approximately 50% of the individual values above and below this value. This suggests that a number of individuals would have serum Ca concentrations below the normal range. Without population variance determinations, you can not really determine how significant this difference is. However, if the herd is experiencing clinical signs consistent with subclinical hypocalcemia, e.g., slow increase in feed intake and milk production, displaced abomasum and ketosis problems among others, this could be interpreted as supportive evidence of the metabolic profile results. Similar interpretive evaluations will need to be made with other metabolites, thus adding some level of subjectivity to this process. This interpretation process underscores the need for integrating results of animal, ration and facilities evaluations with blood parameter results.

Besides comparing individual group means to reference ranges, tracking metabolite concentrations across physiologic groups within a herd may provide useful information. This is assuming there have not been any major feed changes within group rations over a period of time. Cows placed on a protein deficient dry cow ration will initially show low BUN and normal albumin concentrations. By the close-up dry period, BUN may increase slightly as a result of muscle protein catabolism and albumin decreases. This may continue into the fresh cow group resulting in very low albumin concentrations. The author usually can associate a high prevalence of nonspecific infectious and metabolic clinical problems in herds with fresh cows having albumin concentrations at or below 2.5 mg/dL. These poor doing fresh cows may be the result of a compromised immune system unable to accommodate the disease challenges of parturition and early lactation. Changes in group means are correlated with observed clinical signs, BCS changes and ration evaluations to come up with some interpretation and recommendations. So far the author's experiences with this tool have been rewarding and diagnostic. However, more data will need to be accumulated before specific diagnostic parameters can be correlated with specific clinical manifestations.

Confounding Variables to Interpretation

A variety of factors may confound our ability to directly interpret information obtained from a metabolic profile (Table 4). A lack of understanding of underlying physiologic mechanisms can cloud our interpretation of metabolic data. In the CMP, a single static blood metabolite concentration is being used to assess a dynamic physiologic system. What we really need to measure is input-output flux of a specific metabolite. For example, serum magnesium (Mg) concentrations are highly correlated with the presence of clinical signs. However, serum Mg concentrations are not highly predictive for indicating possible impending deficiency disease. Serum Mg concentrations are maintained via dietary intake and renal excretion. If intake is reduced, urinary excretion of Mg will be appropriately reduced to compensate and maintain serum Mg concentrations. In this particular case, urine Mg concentration is a better indicator of deficient Mg intake and impending deficiency disease.

Table 4.

Influencing Factors on Metabolic Profiles

- Homeostatic regulation
- Homeorrhetic regulation - Physiologic states
- Metabolic status
- Diurnal, seasonal variation
- Sample handling - hemolysis
- Disease presence
- Stage of deficiency disease process
- Dietary intake - bioavailability

Nutrients which are homoeostatically regulated, i.e., calcium, sodium, glucose and to some extent phosphorus and potassium, will not show dramatic changes in their blood concentrations over a wide range of dietary situations. This then makes them poor indicators of metabolic status, unless the homeostatic mechanism is deranged. Sampling cows at points in time where they are metabolically stressed, i.e., just prior to and following calving, could potentially result in identifying cows which are more prone to metabolic disease problems. Other metabolites respond slowly to changes as a result of their long half-life, i.e., albumin, total protein, hemoglobin, muscle and liver enzymes. Urea nitrogen concentrations are influenced by a wide variety of interrelated parameters including: dietary protein intake and rumen degradability; dietary amino acid composition; protein intake relative to requirement; liver and kidney function; muscle tissue breakdown and dietary carbohydrate amount and rumen degradability.[7]

For trace minerals, blood or serum concentrations are buffered from acute changes as a result of dietary problems through mobilization of storage mineral, usually from the liver. Four continuous phases, i.e., depletion, deficiency, dysfunction and disease, have been

described to explain the progression of trace mineral deficiency disease.[22] This suggests that liver trace mineral status may be a better indicator of dietary adequacy, whereas measurement of a mineral-specific enzyme activity better reflects the presence of overt clinical deficiency disease compared to blood concentrations. Many trace mineral concentrations in blood are influenced by disease. Bacterial infections induce sequestering of iron and zinc and elevation of copper. This could certainly confound interpretation of blood mineral status. As we come to better understand the factors which affect metabolites, we can adjust and better assess nutritional status. Using strict guidelines as currently suggested without appropriate physiologic interpretation, there will be much overlap between what would be considered normal and abnormal herd profiles.

Energy Balance

Energy balance is by far one of the most critical nutritional factors impacting on animal health, lactational and reproductive performance. Traditionally we have monitored changes in energy balance via body weight and condition changes over time. This procedure, however, may not be a sensitive enough tool when dealing with the transition cow. There is no doubt of the functional role of assessing body condition score changes with lactational performance. Another parameter which might be useful is ketone body concentrations. At present, measurement of 8-hydroxybutyrate (BOHB) concentrations is commonly used. However, BOHB concentrations may not be sensitive enough and can come from other dietary sources. Measurement of blood acetoacetate concentrations would be preferred; however, this is not a feasible test for routine field usage as a result of the rapid deterioration of acetoacetate. A traditional research procedure has recently received much interest in the clinical field. This is measurement of nonesterified fatty acids (NEFA) as a determination of energy balance. Many research studies have shown good correlations between energy balance and serum NEFA concentrations. Serum NEFAs are the result of adipose tissue catabolism of triglycerides and there is no other dietary source. Circulating NEFAs are then absorbed by the liver and other tissues for metabolism to energy. Concentration of NEFA then directly reflects the amount of adipose tissue catabolism taking place. The basis for NEFA and cholesterol recommendations comes from studies and observations associating higher NEFA and lower cholesterol concentrations in cows with higher incidence of periparturient metabolic disease.[23,24] Total cholesterol and NEFA reference values are based on data from Michigan State University Clinical Nutrition Laboratory (Table 5). Serum NEFA concentrations seem to be more sensitive to energy balance changes than body condition scoring in transition cow situations.

Table 5. Suggested serum values for total cholesterol and nonesterified fatty acids (NEFA) in the periparturient dairy cow.[1]

Serum Metabolite	Early Dry	Close-up Dry	Fresh Cow
Total Cholesterol, mg/dL	> 80	> 75	> 100
NEFA, mEq/L	< 0.325	< 0.40	< 0.8

[1]Michigan State University Clinical Nutrition Laboratory, Dr. T.H. Herdt, personal communication.

Protein Evaluation

Assessing protein status is a bit more difficult than energy balance. At present there is no single metabolite which can be measured which directly reflects protein status. As a result, a combination of metabolite parameters need to be utilized, including BUN, creatinine, total protein, albumin and Ck. Creatinine is used to assess renal function and its impact on BUN values. Total protein and albumin reflect availability of protein and their concentrations decline in the face of protein deficiency. However, this occurs over a period of time. Albumin has a fairly short half-life and can reflect protein deficiency problems over a period of a month or two. Creatine kinase is released from muscle when it is catabolized or injured. In most dietary protein deficiency situations, BUN values will be low (<10 mg/dL) with normal albumin concentration (>3.5 gm/dL) in the early dry cows. Close-up dry cows will have low to moderate BUN, lower albumin and elevated Ck values. Fresh cows generally have low BUNs and low albumin (<2.5 gm/dL). These fresh cows seem to fail to properly respond to any disease insult. Cows can die from metritis, mastitis, foot rot and anything else without antibiotic therapy. My interpretation of this situation is that there are no amino acids available to support the immune system and it fails, predisposing the animal to any bug which comes along.

My clinical experience supports this assessment of the role of protein in fresh cow problems. In other situations related to dietary protein deficiency, a high prevalence of retained placentas, metritis, ketosis and eventually displaced abomasums can be seen in the fresh cows. This may be associated with interrelated issues of energy balance and liver function. Recent research work with prepartum protein supplementation has suggested improved energy balance status and liver function.[25,26] These results may relate back to the critical role of amino acids in fetal nutrition and the physiologic processes in place to ensure adequate fetal nutrient supply at the expense of the dam. Work by McNeil *et al.* shows loss of maternal muscle protein during late pregnancy occurred even when ewes were fed

diets containing NRC recommended crude protein levels.[27] **These data support the empirical clinical impressions that we may be underestimating late pregnancy protein requirements for dairy cattle.[28] Clearly further work is needed in this area.**

Liver Function Evaluation

We are all too familiar with the process of fatty infiltration of the liver in the transition cow. Much has been written on the negative role of excessive fatty infiltration and incidence of periparturient disease. Fatty infiltration of the liver is a natural process for the dairy cow transitioning into lactation, but it must be under control. Liver function can be assessed through a variety of enzyme (GGT, AST) Unfortunately, elevations in any of these parameters does not mean anything more than some insult has occurred to the liver. Bilirubin values are most specific to bile flow problems than overt hepatocyte damage. These enzyme values need to be interpreted in conjunction with total cholesterol and NEFA results.

As described above, NEFA are released into the circulation as a direct result of adipose tissue catabolism. The liver takes up NEFA in direct relationship with their concentration in blood. Once in the liver, NEFAs can either be partially metabolized to ketone bodies and distributed to other tissues for energy metabolism or they can be used to synthesize triglycerides (fat). High NEFA values result in either elevated ketones or triglyceride production by the liver. Fat in the liver has two potential options, remain in the hepatocyte and initiate hepatic lipidosis (fatty liver) or be transported out of the liver. In order for fat to be transported out of the liver, protein is required. Fat is transported in blood in compounds termed lipoproteins; this is the only way they are soluble in blood. The lipoprotein structure which transports fat from the liver is called very low density lipoprotein (VLDL). Associated with fat in the VLDL structure is a substantial amount of cholesterol. Therefore, total serum cholesterol indirectly measures the presence of VLDL in blood and consequently measures the liver's ability to produce VLDL. If VLDL production is compromised, fatty infiltration will ensue. Therefore the values presented in Table 5, represent total cholesterol values which characterize conditions in which VLDL production is limited and fatty infiltration is probable. Some investigators have suggested assessing the NEFA to cholesterol ratio for this reason.[23]

Macromineral Evaluation

The macrominerals Ca, P, K, Mg, Na, Cl and S are of extreme interest as to their status relative to their role in milk fever, alert downer cows and weak cow syndrome. Unfortunately, most of these minerals are tightly regulated in the body through a variety of homeostatic processes. Blood concentrations of macrominerals are not reflective at all of dietary status when the homeostatic system is functioning properly. Phosphorus, K, Mg and S are those macrominerals in which blood concentrations are somewhat sensitive to dietary intake. Therefore macromineral blood concentrations will need to be carefully interpreted in light of whether or not the homeostatic system is in proper operation.

In lieu of directly measuring macromineral blood concentration, measurements of parameters directly related to the functioning and responsiveness of the homeostatic regulatory system may offer some insight as to nutritional status. This may be accomplished through measurements of excretion patterns, target tissue responses and homeostatic regulators. As described above, urine magnesium excretion is a more sensitive indicator of potential magnesium deficiency problems than blood concentrations. Relative to Ca, measures of urinary excretion, bone remodeling (blood hydroxyproline concentration) and blood parathormone concentration may be more sensitive to body Ca status. Unfortunately, none of these tests are routinely available at present. As for K and Na, research has focused on concentrations and their ratio to each other in saliva as sensitive measures of K and Na status. Further work to improve the field use of these measures is needed.

Micromineral and Vitamin Evaluation

Assessment of trace mineral and fat-soluble vitamin status is routinely completed using direct blood concentration measurements. The question to ask is whether or not there is a predictive relationship between tissue or blood trace mineral or fat-soluble vitamin concentrations and presence of nutrient-specific deficiency disease. On the surface one would have to say yes because we can document low nutrient concentrations in the presence of disease signs. The question really becomes one of how predictive mineral and vitamin concentrations are and which ones are the best indicators. To understand the issue here we need to appreciate that trace minerals and fat-soluble vitamins are not in a single large pool in the body, but are distributed into a number of different pools which have different functions and availabilities.[22] The different nutrient pools described include a storage, transport and biochemical function pools. As a result of the storage capacity for trace minerals and fat-soluble vitamins in the liver, moderate dietary deficiencies or short-term severe deficiencies can be overcome without any effect on the critical biochemical functions performed by the element in question. If the dietary insult is severe or prolonged enough to drain the storage pool, then some effects might

be seen in the transport pool. Finally, when the transport pool has been compromised, the biochemical function pool will be tapped into. It is only when the biochemical function pool reaches a critically low level that we see the overt clinical deficiency disease we learned about in textbooks. Before we reach the clinical disease stage, we will see problems associated with subclinical disease including increased disease susceptibility as a result of compromised immune function. This is the bulk of the trace mineral and fat-soluble vitamin deficiency disease problems.

The next issue to address is the ability of the chosen marker to be measured and its relationship to changes in one or more of these trace mineral or fat-soluble vitamin pools. Most of our markers currently being used are element concentrations in either whole blood or serum. These probably reflect the transport pool and not necessarily the biochemical function pool. As a result they may not be higher correlated with the presence of clinical disease. A good example here is serum copper concentrations. Unless serum copper is a critically low value, it has no significant predictive value in assessing potential for copper deficiency disease. Another example is the debate between serum and whole blood selenium values. Serum selenium values represent the transport pool and are very sensitive to dietary changes and liver mobilization. On the other hand, whole blood selenium values represent both transport and a portion of the biochemical function pools. This measure is somewhat less sensitive to dietary changes as a result of the greater proportion of whole blood selenium being present as the erythrocyte enzyme glutathione peroxidase. If one was to assess a potential response to a dietary change, serum selenium values would respond within a day or two while whole blood may take a month or more to show a significant change. This could dramatically impact your interpretation of the dietary response.

Liver mineral concentrations are good markers for the storage pool; however, they are not always highly associated with the presence of disease, although, liver mineral concentrations may give us some insight into the adequacy of the mineral program and the potential for disease problems. One additional avenue here is the assessment of mineral status in fetal and neonatal animals. Research has shown that the fetus can concentrate trace minerals in its liver and therefore, comparison to adult values is inappropriate. Secondly, fetal liver has a lower dry matter content than the maternal liver further substantiating the inability for direct comparison. Databases determining normal trace mineral concentrations in the fetal and neonatal liver need to be developed. Obviously we are a long way away from accurately predicting the potential presence of trace mineral deficiency disease problems with our current methodologies. **A number of more predictive markers for specific nutrient pools need to be identified. More research is needed and is being accomplished in this area.**

Summary

Traditional metabolic profiling of the dairy herd resulted in tremendous financial investment with subsequent unsatisfactory results in many situations. A variety of factors are responsible for individual and herd variation in blood metabolite concentrations confounding interpretation. In addition, the cow has an exquisite system of checks and balances which maintains normal physiologic function within a wide array of dietary and environment insults. As a result of these physiologic regulatory mechanisms, simple blood concentration analysis has not been highly rewarding in accurately assessing nutritional and fertility status. A new approach to metabolic profiling of pooling larger sample numbers, specific animal selection relative to physiologic state and stage of lactation has been examined in an effort to better interpret serum metabolite concentrations on a herd basis. Most importantly it must be remembered that metabolic profiles are almost useless without being coupled with animal and facility evaluations, body condition scoring and ration evaluation. The combination used within a team approach can be an extremely useful diagnostic tool in nutritional evaluations of the dairy herd. It is only when the whole picture is evaluated will the use of metabolic profiles produce useful diagnostic information.

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