A GENETIC EVALUATION OF SOME SERUM AND MILK PRODUCTION TRAITS OF DAIRY CATTLE

by

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We accept this thesis as conforming
to the required standard.

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ABSTRACT

This study was initiated to evaluate the genetic aspects of certain serum constituents and milk production traits of dairy cattle. The population under study was located on commercial dairy farms in the upper Fraser Valley region of British Columbia. Serum samples from 545 animals were used in the analysis. This represented 27 sire groups with approximately 20 samples per group.

Serum constituents analysed were calcium, inorganic phosphate, glucose, BUN, uric acid, cholesterol, total protein, albumin, bilirubin, alkaline phosphatase, SGOT, creatinine, triglycerides, sodium, potassium, chloride, bicarbonate, thyroxine and amylase. Production traits studied were milk, milk fat and milk protein for both first lactation and the lactation in progress at time of serum sampling.

Several effects were recognized as having a potentially significant influence on the traits under study. Herd effects were expected to be significant for all traits. Seasonal effects, estimated by sampling in summer and winter seasons, were considered as potentially important. An interaction between herds and seasons was deemed important and included in adjustments for all traits. The covariables used in the analyses were unique to the serum and production traits. Age at lactation start and stage of lactation at time of bleeding were the covariables appropriate for serum traits, while age at lactation start and length of lactation were appropriate

covariables for the production traits.

All effects were evaluated by least squares techniques. The traits under study were adjusted by the least squares constants with the resulting corrected data subjected to the genetic analysis.

The traits that were discerned to have a significant heritability included all the production traits and the following serum constituents: creatinine (0.32), alkaline phosphatase (0.30), amylase (0.20), potassium (0.13), and albumin (0.08). SGOT and BUN were also deemed of interest with heritabilities of 0.05 and 0.05, respectively.

Genetic correlations that existed among the traits were also evaluated. The evaluation of these correlations was carried out in light of the magnitude of the correlation and the relative magnitude of the associated standard errors.

The genetic correlations judged reliable were as follows: creatinine correlated highly and negatively with both milk fat and milk protein for both first and current lactations. Alkaline phosphatase correlated highly and negatively with all production traits for both lactations. Potassium and albumin exhibited reliable, positive correlations with milk production alone.

Genetic correlations were also examined among serum traits in order to elucidate common underlying genotypes. SGOT exhibited a positive correlation with amylase, potassium and albumin, and a negative correlation with alkaline phosphatase. BUN was negatively correlated with amylase and positively

correlated with albumin. Creatinine was correlated positively with alkaline phosphatase, and amylase. Alkaline phosphatase correlated negatively with amylase and potassium. Potassium was positively correlated with albumin.

This study estimated and tabulated the genetic parameters involving some serum and production traits in dairy cattle. Further, it reduced the total number of serum constituents to a subset which demonstrated a genetic component or a genetic involvement in other traits.

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INTRODUCTION

The selection of dairy cattle for the production of milk, milk fat and milk protein has received much attention in recent history. Selection has been intense and researchers have refined selection models and techniques to such a degree that conventional selection procedures are not likely to produce the dramatic improvements achieved in the past (Robertson 1966). In order that progress continue, researchers must explore areas which exploit the restrictions present in the breeding of dairy cattle. These restrictions include the sex-limited nature of the traits, the cost of raising replacement females to an age where the traits are measureable, and the initial selection and cost of raising replacement males. These factors effectively limit the number of animals tested and increase the generation interval, thus limiting selection response per unit time. If selection procedures could overcome these restrictions, it would represent an improvement in genetic and economic terms.

Selection for traits that are correlated with the milk production traits and are available early in life represents one method of overcoming these restrictions. Traits measurable in the blood are among the more promising in this repect. Blood is the intermediary fluid between the mammary system and the rest of the animal. As such, it contains input compounds to the milk synthesis process as well as some compounds superfluous to the process. In addition, blood contains other compounds which are

indicative of general body condition or of the animal's genetic makeup. The latter has been studied by examining the relationships between various polymorphic traits and milk production traits. Some polymorphic systems studied were red blood cell antibody reaction, serum transferrin, and enzyme types. Pirchner (1969) reviewed these relationships and noted the strongest relationships existed between certain blood type alleles and milk fat percentage, yet these relationships accounted for less than ten percent of the genetic variance of milk fat percentage.

Other polymorphic systems have not proven to be highly correlated with milk production. In addition to this failing, there are other intrinsic difficulties associated with polymorphic systems. By definition, they are systems that are composed of a small number of alleles at one or a few loci. Assuming gene action to be additive, selection would cause a trend towards homozygosity in a few generations at the loci involved. The attainment of homozygosity will result in no further response to selection. Also, due to the pleiotropy of gene effect, homozygosity may result in a decrease in performance in other traits, for example, fitness and reproductive performance. There are discrepancies in the statistical and biological interpretation of correlating a system of finite classification (polymorphism) with a trait that is continuously variable (the milk production traits). If the polymorphic system does have an input into the continuously

variable trait, it must be small in relation to the factors which give the trait its continuous distribution. These other factors could be systematic or environmental in nature. The systematic factors offer an area for further study. This present study used traits that were considered to be continuous in their distribution and not based primarily on a polymorphic system.

There are many compounds in the blood that are continuously variable and relevant to milk synthesis. These compounds can be roughly grouped into those forming a material portion of milk, those extracted by the mammary cells and synthesized into milk components, and those which do not form a material part of milk but which mediate some of the biochemical pathways involved in milk synthesis.

Besides the constraints of relevance to milk synthesis, there are other considerations when selecting which blood constituents to measure. Ease and cost of measurement must be considered if it is intended to apply the results in a field study approach. Certain constituents may appear more promising in their relationship with production traits, yet cost of measurement may be prohibitive when applied to a large number of animals. For this reason, this present study was based largely on a human biomedical profile which provided an efficient survey of blood constituents that were indicative of a wide range of body functions. To this profile were added several constituents

which were judged to be of particular relevance to milk production or reproductive performance and which had satisfactory analytical techniques available.

If one is to utilize a trait in conventional selection theory, certain genetic parameters must be known. Heritabilities and genetic variances must be reliably estimated for those traits to be used individually. If one desires to use a number of traits simultaneously, or if one desires to relate the change in production traits concurrent with changes in blood constituents, genetic and phenotypic correlations between these traits must also be estimated.

In this present study, estimates of all the above mentioned genetic parameters will be estimated and evaluated in an attempt to present a more complete and comprehensive investigation. It is proposed that some important relationships between the production traits and some subset of the serum constituents measured can be ascertained.

LITERATURE REVIEW

In a particular study one is usually interested in one or a few major effects. The experiment is set up and carried out in order to facilitate the analysis of these effects. In general, there are other factors which are significant but not of prime importance. These secondary effects must also be quantified as accurately as possible and taken into account in the analysis. In this particular study, the main interest was in a sire effect so that heritabilities and correlations could be derived and tested statistically. Beyond the sire effect, many readily identifiable factors affect blood composition. If they can be quantified and interpreted, they deserve a place in the analysis. It is optimal to identify and remove as many significant effects as possible, in order to get a complete understanding of the nature of the blood constituents.

Age has been cited as a factor in governing levels of serum constituents by many researchers. Age may be due to a number of processes that appear or change as the animal matures. Within the range present in this study, age may not be as important since the animals were a more homogeneous group than expected under random conditions. All animals sampled in this study were lactating and therefore the age range of 0-23 months was not represented. The actual age range in this study was 23 months to 157 months, with a concentration in the lower portion of this

range. There still was present, however, the effects of continued growth, the maturing processes and age related stresses.

Serum calcium levels have been reported to be significantly affected by age. Tumbleson et al. (1973b) reported a significant linear decrease in calcium level with age. No significant age effect was reported by Kitchenham et al. (1975) and Kitchenham and Rowlands (1976). This conclusion was based on values obtained over a number of herds.

Inorganic phosphate has been noted to decrease with age. Tumbleson et al. (1973b) reported a significant curvilinear relationship, while Kitchenham et al. (1975) and Kitchenham and Rowlands (1976) termed it a significant linear relationship.

Sodium, potassium and chlorine were not reported to be affected by age, over the range in the present study (Tumbleson et al. 1973b and Kitchenham et al. 1975). Blood urea nitrogen (BUN) followed a similar pattern, that is, significantly affected by age up to two years but stabilizing after that point (Tumbleson et al. 1973b and Kitchenham et al. 1975). The study by Kitchenham and Rowlands (1976) indicated a significant decrease in sodium and urea with age, although the range studied was 1.25 to 12.75 years. This range was wider than our experimental range. Since most of the significance appeared in the 0 - 24 months of age range, the reduced lower limit may account for the significance noted in this study. Furthermore, Kitchenham and Rowlands (1976) noted

that the decrease of sodium with age may have been specific to the herd under study.

Serum proteins have been noted to be affected by age. Tumbleson et al. (1973a) stated that total protein increased with age while alkaline phosphatase decreased with age. Albumin showed no relationship with age. Little (1974) noted an increase of albumin from two through four years of age but no significant relationship thereafter.

Lipid compounds measured in the present study were cholesterol and triglycerides. Arave et al. (1975) indicated an increase in cholesterol concentration with age but again the range differed from that used in the present study. Total lipids were positively correlated with cholesterol (r=0.71). Triglycerides make up a large portion of total serum lipids and it may be expected that triglycerides levels are also affected by age. Tumbleson and Hutcheson (1971) further clarified the situation by stating cholesterol levels increased up to three years of age and decreased thereafter. This decrease after three years of age was speculated to have been caused by selective culling of older animals.

Bilirubin was noted by Tumbleson and Hutcheson (1971) to significantly increase with age. Mylrea and Healy (1968) concurred with this statement.

Thyroxine secretion rate declined with increasing age over the normal productive age range in dairy goats (Flamboe and Reineke 1959).

Kitchenham et al. (1975) noted an increase in serum glucose with age in a study involving a number of herds. Kitchenham and Rowlands (1976), in a within herd study, reported no relationship between glucose and age. Further obscuring this situation was a report by Tumbleson and Hutcheson (1971). This was a within herd study which showed a significant relationship from 0.5 to 5 years of age, with no change thereafter. The range of significance encompasses most of the present study's data, although the lower age limit was again much lower.

Peterson and Waldern (1978) reported significant decreases with age in the level of inorganic phosphorus, BUN, cholesterol, alkaline phosphatase, serum glutamic oxaloacetic transaminase (SGOT) and albumin, and significant increases in total protein and glucose. These were in close agreement with Kitchenham et al. (1975) and Tumbleson et al. (1973a) and (1973b).

Seasonal effects were considered potentially important for the population studied. Blood samples were taken in both summer and winter, and the difference in sampling period was taken as a measure of the seasonal effects. These effects may have included differences in climate, diet and feeding management as major causal factors.

Relatively few of the constituents present in blood serum have been shown to vary seasonally. Sodium was noted to be significantly lower in summer than winter, while urea and

albumin concentrations were higher in summer (Payne et al. 1974). Ross and Halliday (1976) disputed this relationship for albumin, and further stated that calcium was significantly higher in the summer. Urea was found (Payne et al. 1974) to be higher in summer. This may be due to increased protein intake in grass in the summer. Increased dietary protein has been shown by some studies (Little and Manston 1972, Manston et al. 1975, Prewitt et al. 1971) to affect BUN. This effect should be lessened when herds have a consistent feeding program throughout the year.

Ross and Halliday (1976) noted higher summer levels of serum cholesterol, although this was in general conflict with Arave et al. (1975) who noted generally lowered cholesterol levels in summer. Neither study provided strong basis for the argument of significant seasonal effect.

Information on seasonal effects for SGOT and alkaline phosphatase was also limited. In a summer-winter system of bleeding, Roussel and Stallcup (1966) noted a significant seasonal difference for both enzymes. The importance of this result was tempered by the fact that the range of age for the animals (0-2 years) in this study was not the same as in the present study, and by the statement made by Roussel and Stallcup (1966) that, age differences in their study could not be completely separated from seasonal effects.

Two papers (Flamboe and Reineke 1959, Mixner et al. 1962) indicated significant seasonal effects with regards to thyroxine

level in the blood.

Herd effects were noted to account for the largest single portion of the variablity associated with many blood constituents (Hewett 1974, Payne et al. 1974, Rowlands and Manston 1976). This was consistent with the observation that herd differences are comprised of a number of smaller effects, notably feeding, management and the microenvironment associated with the farm.

Virtually all blood constituents involved in the present study have been reported in the literature to be significantly affected by herd. Payne et al. (1973) and (1974) in two studies indicated that all constituents involved in the Compton Metabolic Profile (CMP) were significantly affected by herd. Hewett (1974) concurred with these results. The constituents common to the Compton studies and the present study were glucose, BUN, inorganic phosphate, calcium, sodium, potassium and albumin. Wilson and Dinkel (1968) arrived at the same conclusion regarding the importance of herd effect. Specifically, they noted the significance of herd on the variability of creatinine and alkaline phosphatase.

The herd effect on uric acid, cholesterol, total protein, bilirubin, SGOT, triglycerides, chlorine, bicarbonate, thyroxine and amylase was not well reported in the literature. From the literature reviewed, it was expected herds would be important contributors to the variation in most of the parameters

measured. Moreover, some statistical complications may arise due to interactions between herds and other sources of variation. Payne et al. (1974) indicated that the herd by lactational group interaction was large in comparison to lactational group effect.

Since the stage of lactation at time of sampling is associated with different production stresses and nutritional regimes, it should be taken into account as a source of variation in blood constituent levels. Many studies in the literature included some measure of lactation stage in their analysis. In one of the Compton studies, Rowlands et al. (1975) noted the general situation of significant variability in constituent levels due to stage of lactation in the range of 0-120 days, with little effect beyond this range. Exceptions to this general situation were inorganic phosphate and potassium, both of which showed no significant trend with regards to stage of lactation. More specifically, the results of Rowlands et al. (1975) indicated the following: glucose, BUN, total protein and calcium were lowest at the start of lactation and showed a significant increase thereafter. Sodium decreased as lactation progressed. Albumin showed the strongest relationship with lactation, especially in the early stages. Little (1974) concurred with this positive relationship. Hewett (1974) also looked at effect of lactation on an extended blood profile, using monthly groups as stages of lactation. He noted inorganic

phosphate and potassium were not affected by lactation, and that sodium, total protein and BUN were significantly affected. These results were in agreement with Rowlands et al. (1975). On the other hand, Hewett (1974) reported glucose, calcium and albumin were not significantly affected by lactation. It should be pointed out that Hewett (1974) was considering a much larger range of lactation stage in his studies.

Hewett (1974) also noted that there was no significant effect of stage of lactation on any of three measures of thryoid activity, namely protein bound iodine, total iodine and inorganic iodine. Mixner et al. (1962) noted a significant effect on thyroid secretion as measured by the chemical thyroxine turnover method.

The effect of an animal's genotype on the variability of blood constituent level is of prime importance in the present study. Body metabolites and biological compounds present in body fluids are the result of synthesis of nutrients absorbed from the gut, and or arise as by-products of bodily functions. It is generally accepted that many of these processes are under genetic control, either directly, as in protein synthesis, or indirectly through control of enzyme production. Whether this genetic control was associated with a portion of the observed variability in blood constituent level was under question and has received some attention in the literature.

Studies involving the CMP reported a significant

heritability for many of the constituents tested. Using halfsib correlations, Kithchenham and Rowlands (1976) reported significant heritabilities (±standard error) for BUN (0.77±0.38), albumin (0.47±0.33), total protein (0.50±0.34), calcium (0.46±0.33) and potassium (0.53±0.34). Sodium, glucose and inorganic phosphorus were associated with insignificant heritabilities. The standard errors associated with these heritabilities were quite large, due mainly to the experimental design which resulted in small progeny groups of approximately four animals each. Robertson (1959) stated that small progeny group size will result in high standard errors and thus unreliable estimates. Dam-daughter regressions in the same study by Kitchenham and Rowlands (1976) showed reduced heritability estimates with more reliable standard errors. However, these estimates were significant only for total protein. An earlier study by Rowlands et al. (1974) estimated heritabilities for a number of constituents using halfsib analysis on beef calves of 9-11 weeks of age. After eliminating differences associated with the two regions involved in the study, significant heritabilities were estimated for glucose (0.18±0.16), potassium (0.40±0.23), calcium (0.19±0.17), albumin (0.10±0.13) and inorganic phosphate (0.18±0.16). Nonsignificant heritabilities were estimated for BUN and sodium. This was in rough agreement with the former study, but two points should be made. One is that the second study dealt with animals outside the range of age used in this present study. The other point is

that the study involving the beef calves was of such a design as to minimize the standard errors of estimate and thus were more reliable.

Wilson and Dinkel (1968) reported on a set of constituents which were largely in addition to those present in the CMP. They noted insignificant heritabilities for inorganic phosphate, creatinine and alkaline phosphatase. Bettini et al. (1975) reported heritabilities for a large number of constituents under examination in the present study. They listed significant heritabilities for calcium (0.22), inorganic phosphate (0.21), glucose (0.19), BUN (0.10), uric acid (0.47), cholesterol (0.40), albumin (0.46), bilirubin (0.12), alkaline phosphatase (0.12) and SGOT (0.19). This study used 213 cows in eight daughter groups, a design which should yield reliable esimates. Furtmayr (1975) estimated significant heritabilities for glucose (0.45), but reported insignificant heritabilities for bilirubin, cholesterol and SGOT.

The genetic basis of serum cholesterol level is widely reported. Arave et al. (1975) noted $h^2=0.50$ for dairy cattle in their first lactation. Stufflebeam and Lasley (1969) and Taylor et al. (1966) concurred with this relatively high heritability.

Some studies have attacked the problem of a genetic basis of blood constituent levels by measuring the effect of breed. If this effect is significant, then it may be reasonable to postulate a genetic base and thus a heritable portion of the variability in blood constituent levels. Heyns (1971a) noted

significant breed differences for alkaline phosphatase, potassium, calcium and glucose. Nonsignificant breed differences occurred for albumin, sodium, and inorganic phosphate. Kunkel et al. (1953) noted significant breed differences for alkaline phosphatase although they noted further that repeatability within breed is low, indicating environmental effects overshadow breed effects.

Peterson and Waldern (1978) have estimated repeatablilities for a group of serum constituents basically the same as the ones involved in the present study. Since that study was set up in part to estimate repeatabilities, the results were reliable. The results of this study were of interest insofar as repeatability is an upper limit of heritability. They reported moderate to high repeatabilities for inorganic phosphate (0.194), creatinine (0.514), total protein (0.613), alkaline phosphatase (0.546), SGOT (0.264), glucose (0.205) and albumin (0.246) for animals in lactating, nonpregnant condition. The majority of cows in the present study were in this conditon.

Correlations among levels of blood constituents and between blood constituents and production traits could be of great value in selection of breeding stock and prediction of future performance. The Compton researchers have noted some correlations among the constituents they have studied. Kitchenham and Rowlands (1976), using correlations adjusted for age and breed, noted a significant positive correlation between

calcium and albumin (r=0.33). Payne et al. (1974), using simple correlation coefficients, supported this finding, although it was apparent only in the summer period. They further noted a significant positive correlation between inorganic phosphate and potassium, with r=0.40 in the winter season and r=0.24 in the summer season. In an earlier study, Payne et al. (1973) reported some significant partial correlation coefficients (BUN with inorganic phosphate (0.49) and with albumin (0.42), inorganic phosphate with potassium (0.25 to 0.60)), although their importance was limited by the fact that herd means were used in the calculations. Thus the within herd variability, which was judged to be both large and erratic for these correlations (Payne et al. 1973, Kitchenham et al. 1975), was not taken into account. Rowlands et al. (1974) listed significant correlations of glucose with albumin (0.40), glucose with sodium (0.34), glucose with calcium (0.43), glucose with inorganic phosphate (0.34), albumin with sodium (0.30), albumin with inorganic phosphate (0.39), albumin with calcium (0.60), sodium with inorganic phosphate (0.31) and sodium with calcium (0.31).

Heyns (1971b) reported correlations among several constituents involved in the present study. Several of these were in addition to those constituents studied by the Compton group. A negative correlation between albumin and BUN (r=-0.180) was in agreement with a study previously mentioned. Heyns also presented significant positive

correlations of creatinine with BUN (0.212), albumin with phosphorus (0.397), glucose with alkaline phosphatase (0.290) and with phosphorus (0.192), and alkaline phosphatase with phosphorus (0.179). Negative correlations were reported for glucose with BUN (-0.209) and with creatinine (-0.345).

With regards to the estimation of correlations between levels of blood constituents, it was apparent that most of the studies reported in the literature were not set up primarily to deal with this aspect. Instead, interest in correlations appeared to be initiated after the collection of data in order to expand the scope of the study. To be most worthwhile, a study should be designed to estimate correlations with a minimum of constraints. Furthermore, the design should allow subdivision of this phenotypic correlation into both genetic and environmental components.

Correlations between blood constituents and milk production traits have received some attention in the literature. Payne et al. (1973), using the criterion that an "abnormal" level of a serum constituent is one which is greater than two standard deviations away from the population mean, reported that "disappointing milk quality and yield" was associated with high levels of potassium or with low levels of albumin, calcium or sodium. Of these indications, the relationship with low levels of albumin was much the strongest. These were determined on a herd average basis and thus disregarded individual cow

differences.

Kitchenham et al. (1975) reported significant partial regression coefficients between milk yield and glucose ($b'=-0.082$), BUN ($b'=0.033$) and albumin ($b'=0.0095$), indicating that some degree of relationship exists. Sink et al. (1973) looked at several interrelationships between serum lipids and production traits, but were unable to report any significant correlations between serum cholesterol and any milk production trait.

Two other studies contained extensive lists of correlations between some serum constituents and the milk production traits. Furtmayr (1975), in his Inaugural Dissertation, reported on a study involving 168 Holstein-Friesians. His findings were that milk yield was positively correlated with cholesterol (0.20) and with SGOT (0.33). A negative correlation existed between glucose and milk yield (-0.20). Milk fat percentage was positively correlated with bilirubin (0.28) and negatively correlated with glucose (-0.27) and cholesterol (-0.25). Bondarenko et al. (1976) indicated significant, positive correlations between milk fat percentage and triglycerides, cholesterol and albumin.

MATERIAL AND METHODS

a) Data Collection and Analysis:

The animals sampled in the present study were Holstein-Friesian dairy cattle in commercial dairy herds located in the eastern Fraser Valley region of British Columbia. These 35 co-operating herds were chosen to give as representative a sample as possible with regards to nutrition, management techniques, microenvironments within the area and ability to cclIect production data from a recognized data collection scheme.

Specifically the herds used had the following characteristics. Herd size ranged from 21 to 136 milking cows with a quadrimodal distribution of 40, 51, 56, and 59 cows per herd. Several management and feeding systems were used. Many farms utilized a pasturing system in the summer and most supplemented with stored feeds. Several farms fed conserved forages exclusively throughout the year. Included in the overall classification of stored feed were hay, corn silage, grass silage and grass-legume silage and, on different farms many combinations of these feeds were used. Housing systems included traditional stanchion barns, loose housing and freestall systems, with the latter being most numerous. Milking systems varied accordingly. Bucket and pipeline milking systems were found in stanchion barns, while various parlour systems were associated with loose and freestall housing.

Cow samplings within each herd were carried out in order to facilitate the valid estimation of all sources of variation that were discussed in the literature review. The methods and considerations that were employed in the sampling within herd follow.

Age at lactation start was one consideration in the sampling scheme. Sampling was concentrated on young, lactating animals with relatively few animals of advanced age. This was done for a number of reasons. Firstly, this skewed distribution was representative of the structure of many herds. Secondly, animals in their first lactation would be less heavily selected and less subject to special treatment than older animals that were of known productive ability. Thus, younger animals would yield a sample in keeping with the assumption of randomness.

Stage of lactation, measured as days from lactation start to date of blood sampling, was also a consideration. Samplings were concentrated in the first 120 days of lactation to emphasize the period of greatest production stress. If there were relationships between blood constituents and production traits, it could be expected that these relationships will become more pronounced in periods of stress or marginal intake of nutrients in relation to requirements.

Seasonal effects were estimated by sampling each herd on

two separate occasions. The summer blood sampling was carried out between July 19 and 28, 1976, and the winter sampling was carried out between January 19 and February 5, 1977. These two sampling seasons represented as much difference as possible in climate, nutrition and management. They also represented periods at which the herds would have had adequate time to adjust to any seasonal changes. The same individuals were not sampled in both seasons in order that the considerations of the statistical design and its assumptions could be met.

The sire component of variance was the subject of prime interest in this study. The numbers of animals sampled and the distribution of this sampling were arrived at in part by considering the reliability of the estimates of the genetic parameters that they would produce. Robertson (1959) stated that when estimating heritabilities from sib analysis, small sib group size (less than 10 individuals per group) will result in unacceptably high standard errors and thus unreliable estimates. The standard error of heritabilities, according to Robertson (1959), are minimized when sib groups are between 10 and 40 animals. On this basis, the present study was set up to subdivide the animals sampled into sire groups of desirable size. The total number of animals sampled was 1030 of which 700 were analysed for the serum constituents. Within the animals for which serum samples were analysed, 27 sire groups of 10 or more daughters could be ascertained. The average sire group

size was approximately 20 animals for a total of 545 animals with complete data. This should result in reliable estimates and acceptable standard errors.

Blood was obtained from the jugular vein and collected in vacutainer tubes containing no anticoagulant. These blood samples were allowed to clot and then were centrifuged within 6 hours of collection. Serum was pipetted and the entire sample from one animal split into four subsamples of 2 ml or more. These subsamples were placed immediately into a freezer and stored for approximately six months at -18° C until sent to a biomedical laboratory[1] for analyses.

The serum constituents measured on a Technicon AutoAnalyzer SMA 12/60 were calcium (Ca), inorganic phosphate (PO4), glucose (Gluc), SGOT, cholesterol (Chol), albumin (Alb), BUN, uric acid, alkaline phosphatase (Alk P), bilirubin (Bili), and total protein (T P). Other constituents, which were measured on other equipment, were sodium (Na), potassium (K), creatinine (Creat), amylase (Amyl), triglycerides (Trig), chloride (Cl), bicarbonate (HCO3), and the T4 fraction of thyroxine.

The constituents present in ion form in serum were calcium, sodium, potassium, chloride and bicarbonate. Calcium was determined by a method modified from that of Kessler and Wolfman

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[1]B. C. Biomedical Laboratories, Burnaby, B. C.

(1964). This modification virtually removed any interference from magnesium. The determination was colorimetric and units of measurement were milligrams per decilitre (mg/dl).

Sodium and potassium were determined by spectrophotometric methods using a Beckmann potassium, lithium, sodium flame with an internal lithium standard. Units of measurement for both elements were millieguivalents per litre (meq/l).

Chloride and bicarbonate were measured in the serum by the method outlined by Kenny and Cheng (1972). Briefly, this was a simultaneous determination with both constituents being determined by cclorimetry on a Beckmann chloride carbon dioxide analyzer. Units of measurement were meq/l.

Protein and compounds related to protein metabolism that were measured in this study were total protein, albumin, BUN, uric acid, bilirubin and creatinine. Total protein was determined by a modified biuret reaction as described by Skeggs and Hochstrasser (1964). Determination was colorimetric and units were grams of total protein per decilitre (g/dl).

Albumin was analysed by the method of Doumas *et al.* (1971) in which a colorimetric reaction with bromcresol green was used. It was noted this technique was free from interference with some other serum constituents, most notably bilirubin. Units of measurement were g/dl.

BUN determination utilized the method reported by Marsh *et al.* (1965). This method entailed the reaction of urea with

diacetyl-monoxine in the presence of compounds that enhance the color development. The advantage of this method over earlier methods was in its ability to utilize reactions of low acidity. Determination was colorimetric and units were mg/dl.

The method of analysis of uric acid was an automated version of the method set out by Sobrinho-Simeos (1965). It was based on reduction of a phosphotungstate complex which resulted in stable colour development in the presence of stabilizing and color intensifying agents. Determination was colorimetric and units were mg/dl.

Total bilirubin was estimated by the method of Jendrassik and Grof as adapted by Gambino and Schreiber (1964). Serum bilirubin was reacted with diazotized sulfanilic acid in the presence of a caffeine-sodium benzoate reagent to give differential colorimetry between a sample and a blank reaction, with units of mg/dl.

Creatinine was measured by the Jaffe Method which invclved colorimetric determination of the reaction between creatinine and alkaline picrate. Units of measurement were mg/dl.

Enzymes measured in the serum were SGOT, alkaline phosphatase and amylase. SGOT was determined by the method of Morganstern et al. (1966). Oxaloacetic acid was generated from SGOT, dialyzed and coupled with Fast Ponceau L, a diazonium salt. This method eliminated the need for running a concurrent control and correlated well with other determination techniques.

This was a colorimetric measurement, with units of milliInternational Units per millilitre (mU/ml).

Alkaline phosphatase was measured by use of enzymatic hydrolysis of p-nitrophenyl phosphate and subsequent color development. This method, as reported by Morganstern et al. (1965), eliminated interference by bilirubin and the need for correction with a blank standard. Units of measurement were in mU/ml.

The measurement of amylase level in serum was accomplished by the method outlined by Rinderknecht et al. (1971). This method was based on release of soluble starch fragments due to reaction of amylase with an insoluble starch labelled with Remazolbrilliant Blue. Determination was colorimetric and units were Somogyi units.

The two lipid constituents measured were triglycerides and cholesterol. Triglyceride determination was by the method of Buccio and David (1973) and involved enzymatic hydrolysis of triglycerides by a microbial lipase and a protease. The hydrolysis procedure was subsequently coupled to an enzymatic determination of glycerol through use of absorbance in a spectrophotometer. This determination was stated to be specific for triglycerides. Units of measurement were mg/dl.

Cholesterol was analysed by the method of Levine et al. (1967). This was a modification of an earlier method and involved the reaction of cholesterol with glacial acetic acid,

acetic anhydride and sulfuric acid. Determination was by colorimetry and units were mg/dl.

Other compounds which were measured in the serum were glucose, inorganic phosphate and thyroxine. Glucose was determined by the method of Bondar and Mead (1974). The determination reaction involved a spectrophotometric measurement corrected by a blank determination. Units were mg/dl.

Inorganic phosphate measurement was based on formation of phosphomolybdic acid and subsequent reduction by stannous chloride-hydrazine. This method was described by Hurst (1964). It was a colorimetric technique with units of mg/dl.

The method described by Cheung and Slaunwhite (1976) was used in the determination of the T4 fraction of thyroxine. This method utilized radioimmunoassay techniques and measured T4 in units of micrograms per decilitre.

The milk production data used in this study was obtained from two sources. Data from herds on the provincial Dairy Herd Improvement (DHI) program was obtained from the central data location in Victoria, B. C. The particular data collected included 305 day lactation yields of milk, milk fat and milk protein for both the first lactation and the lactation in progress at the time of bleeding. Since the number of herds on the federal Record of Performance (ROP) program was small, data were collected from these herds during farm visits. Type of

data collected from ROP herds was identical to that from DHI herds with the exception that milk protein was not available from the ROP herds. Serum data were available for 701 animals, but only 545 cows were used in the final analyses. The reason for this reduction was the inability to match some cow identification numbers recorded at the time of blood sampling with those present at the central data locations, deletion of outliers, and maintenance of optimal sib group size.

b) Statistical Models:

There were several effects to be accounted for in this analysis with the sire effect being of prime importance. Since all effects and covariables were not common to both production and serum traits, each dependent variable was analysed by the appropriate model to eliminate unique effects, and then joint analyses were performed on adjusted data to obtain estimates of the various genetic parameters. Adjustment factors were obtained using least squares constants from the appropriate analyses.

For the milk production traits, the model on which adjustments were based was as follows:

$$Yijk = u + Hi + Sj + HSij + bAijk + cLijk + \epsilon ijk$$

where

u = overall mean common to all samples.

Hi = the effect of the i-th herd.

Sj = the effect of the j-th season of freshening.

HSij = the effect of the i-th herd specific to the j-th season.

Aijk = the covariable age (mo.) to appropriate lactation start.

b = the coefficient associated with Aijk.

Lijk = the covariable lactation length (days).

c = the coefficient associated with Lijk.

ϵijk = the unexplained environmental deviations associated with individual samples.

For the serum traits, the model on which adjustments were based was as follows:

$$Yijk = u + Hi + Sj + HSij + bAijk + cLijk + \epsilon ijk$$

where

u = overall mean common to all samples.

Hi = the effect of the i-th herd.

Sj = the effect of the j-th season of bleeding.

HSij = the effect of the i-th herd specific to the j-th season.

Aijk = the covariable age (mo.) to start of lactation in which bleeding occurred.

b = the coefficient associated with Aijk.

Lijk = the covariable days from lactation start to bleeding date (stage of lactation).

c = the coefficient associated with Lijk.

ϵijk = the unexplained environmental deviations associated with individual samples.

The effects of herd, season and herd x season interaction were treated as fixed effects. All effects were assumed to be normally distributed and independent with expectations equal to zero. Constants were estimated and significance tested for all appropriate terms in the above models by use of least squares analysis. The raw data were adjusted for all appropriate adjustment terms, regardless of significance. This was done recognizing that an insignificant adjustment term would neither alter the data significantly nor introduce bias.

Using the adjusted data, the sire component of variance was estimated from the following model

$$Yij = u + Si + \epsilon ij$$

where

u = overall mean common to all samples.

Si = the effect of the i-th sire.

ϵij = the unexplained environmental deviations associated with individual samples.

Sire was treated as a random effect and was assumed to be normally distributed with expectation equal to zero.

c) Estimation of Variance Components:

Referring to Table I, this present study utilized the following components of variance (with definitions as outlined

Table I. Analysis of variance (ANOV) table and expected mean squares (EMS) for the experimental models.

Sources of variation	d.f.	EMS
Least Square Correction		
Covariables	2	
Age (months)	1	
Stage of lactation[1] (days)	1	
Herd	d.f.h	$\sigma^2e + k4\theta^2h$
Season	d.f.se	$\sigma^2e + k3\theta^2se$
H x S	d.f.hs	$\sigma^2e + k2\theta^2hs$
Genetic		
Sires	d.f.sires	$\sigma^2e + k1\theta^2s$
Individual/Sires	d.f.e	σ^2e

where $k1 = (N.. - (\sum Ni.)^2/N..)/d.f.sires$

[1] For production traits, this becomes length of lactation.

by Falconer (1960):

$\sigma^2 e$ = the component of variance associated with individuals within sire groups. This component of variance contains 3/4 of the additive genetic variance (V(A)) and all the environmental variance (V(E)). Nonadditive genetic variances were assumed to be negligible in this system.

$\sigma^2 s$ = the component of variance associated with differences among sire groups. This was assumed to lead to an estimate of 1/4 V(A). Variances associated with additive by additive interactions were assumed to be negligible in this system.

The variance components of interest in this study were V(A), V(E) and the phenotypic variance V(P). These were calculated using the following relationships:

$$V(A) = 4\sigma^2 s$$

$$V(E) = \sigma^2 e - 3\sigma^2 s$$

$$V(P) = V(A) + V(E)$$

d) Variance of Variance Components:

Variances of variance components were calculated by the formulae given by Searle (1971a). For the components estimated in this study, the appropriate sampling variances were:

1) $Var(V(A)) = 16 Var(\sigma^2 s)$

where

$$Var(\sigma^2 s) = 2\sigma^4 e N..^2 (N..-1)(A-1)/(N..-A)(N..^2-S2)^2 + (4\sigma^2 e \sigma^2 s N..)/(N..^2-S2) + 2\sigma^4 s (N..^2 S2 + S2^2 - 2N..S3)/(N..^2 - S2)^2$$

and

$N.. = \sum Ni.$

$S2 = \sum Ni.^2$

$S3 = \sum Ni.^3$

A = the number of sire groups

$Ni.$ = the number of samples in the i-th sire group

2) $Var(V(E)) = Var(\sigma^2 e) + 9Var(\sigma^2 s) - 6cov(\sigma^2 e \sigma^2 s)$

where

$Var(\sigma^2 e) = 2\sigma^4 e/(N..-A)$

$cov(\sigma^2 e \sigma^2 s) = -N..(A-1)\,Var(\sigma^2 e)/(N..^2 - S2)$

3) $Var(V(P)) = Var(V(A) + V(E))$

$= Var(\sigma^2 s + \sigma^2 e)$

$= Var(\sigma^2 s) + Var(\sigma^2 e) + 2cov(\sigma^2 e \sigma^2 s)$

e) Heritability Estimates:

Heritability estimates were derived as described by Falconer (1960). Intraclass correlations (t) between halfsib groups were employed, making use of the factors comprising the sire and environmental components of variance. In general,

$h^2 = 4t = V(A)/(V(A) + V(E)) = 4\sigma^2 s/(\sigma^2 e + \sigma^2 s)$

f) Variance of Heritability Estimates:

Variances of the heritability estimates were calculated making use of the relationship between heritability and the intraclass correlation between halfsib groups. The sampling variance of the intraclass correlation for halfsib analysis was a modified version of that reported by Robertson (1959).

$$Var(h^2) = 16Var(t)$$
$$= 32[1+(k1-1)t]^2(1-t)^2/k1(k1-1)(A-1)$$

g) Correlation Estimates:

Estimates of genetic, environmental and phenotypic correlations for each pair of traits measured in this study were tabulated using the following formula:

$$r(XaXb) = cov(XaXb)/\sigma Xa\sigma Xb$$

where

$r(XaXb)$ = the appropriate correlation between traits Xa and Xb ($a \neq b$)

σXa = the appropriate standard deviation of the a-th trait

σXb = the appropriate standard deviation of the b-th trait

and where $cov(XaXb)$ was the solution of a rearrangement of the following general formula:

$$Var(Xa+Xb) = \sigma^2 Xa + \sigma^2 Xb + 2cov(XaXb)$$

h) Variance of Correlation Estimates:

Variances of the correlations calculated in this study were estimated by the approximation given by Scheinberg (1966).

$$\text{Est. Var}(rXaXb) = 2ur^2(XaXb)/k1^2\{(sV^2a/\text{d.f.sires} + w^2v^2a/\text{d.f.e})/4\sigma^4a + (sV^2b/\text{d.f.sires} + w^2v^2b/\text{d.f.e})/4\sigma^4b + s[(VaVb + V^2ab)/\text{d.f.sires} + w^2(vavb + v^2ab)/\text{d.f.e})]/2\text{cov}^2(XaXb) - (sVaVab/\text{d.f.sires} + w^2vavab/\text{d.f.e})/\sigma^2a\text{cov}(XaXb) - (sVbVab/\text{d.f.sires} + w^2vbvab/\text{d.f.e})/\sigma^2b\text{cov}(XaXb) + (sV^2ab/\text{d.f.sires} + w^2v^2ab/\text{d.f.e})/2\sigma^2a\sigma^2b\}$$

where

Va = sire mean square associated with trait a

Vb = sire mean square associated with trait b

Vab = sire mean covariance for traits a and b

va = residual mean square associated with trait a

vb = residual mean square associated with trait b

vab = residual mean covariance for traits a and b

and

u = 16, 1 and 1 for the genetic, environmental and phenotypic correlations respectively

s = 1, 9 and 9 for the genetic, environmental and phenotypic correlations respectively

w = 1, k1+3 and k1-3 for the genetic, environmental and phenotypic correlations respectively

RESULTS AND DISCUSSION

Before subjecting the data to analyses for the genetic parameters, least squares adjustments for identifiable, systematic environmental effects were undertaken. The effects of herd, season, herd by season interaction, and two covariables were taken into account. Age at lactation start, one of the covariables, was common to all traits. For first lactation records, this was the age to start of first lactation. For both current production and serum traits, this covariable was the age at start of the lactation in which serum sampling took place. The second covariable was length of lactation in the case of the milk production traits and stage of lactation at time of bleeding in the case of the serum constituents. The actual least squares constants associated with herd and the interaction term were of little interest since no specific inferences were to be drawn from these effects. However, the least squares constants associated with the two seasons and the two covariables allowed us to state in which season the level of a trait was higher or how the level was changing with respect to the covariable. For this reason, an abridged table of least squares constants is presented in Table II. This table shows the trends for the seasons and the covariables in a form more concise than written text.

The most noticeable of these least squares constants were

Table II. Least squares constants associated with summer and winter seascns and with the covariables.

Trait	Season		Covariables		
	Summer	Winter	Age	Length	Stage
Milk1	-214.4	214.4	41.98	28.56	na
Fat 1	-6.351	6.351	1.605	1.250	na
Prot1	-5.454	5.454	1.549	.9522	na
MilkC	-195.3	195.3	22.36	34.26	na
Fat C	-5.708	5.708	.8728	1.272	na
ProtC	-4.556	4.556	.6318	1.127	na
Ca	.0379	-.0379	-.0021	na	.0005
PO4	.0139	-.0139	-.0104	na	.0011
Gluc	-2.090	2.090	.0194	na	.0140
BUN	.1333	-.1333	-.0178	na	.0098
Uric	.1130	-.1130	-.0001	na	-.0004
Chol	-6.949	6.949	-.1069	na	.0523
T P	.0406	-.0406	.0100	na	-.0006
Alb	-.2449	.2449	.0004	na	.0003
Bili	-.0049	.0049	.0002	na	-.0002
Alk P	-.6511	.6511	-.1720	na	.0187
SGOT	-.4055	.4055	-.0543	na	.0536
Creat	-.0693	.0693	-.0004	na	-.0000
Trig	.0890	-.0890	-.0122	na	.0030
Na	-.8073	.8073	-.0154	na	.0016
K	.0167	-.0167	-.0009	na	-.0000
Cl	.6487	-.6487	-.0071	na	.0050
HCO3	-.9059	.9059	-.0044	na	-.0019
T4	-.2352	.2352	-.0087	na	.0047
Amyl	-8.706	8.706	.5502	na	.0122

those associated with age in the first and the current lactations. The least squares constants for all three measures of production were considerably higher for first lactation compared to those for current lactation. This indicated that, over the smaller range of age present in first lactations (23-36 months), age had a more pronounced effect which was reflected in the higher associated constants. Current lactation production, with an age range of 23-157 months, did not show as strong a relationship and consequently had much lower constants for age.

The coefficient of determination (R^2) for the total model is a measure of the portion of total variability accounted for by the environmental effects listed above. For the traits examined in this study, the total R^2 ranged from 0.208 for alkaline phosphatase to 0.704 for albumin. This indicated the relatively small importance of the systematic, environmental effects utilized in this study in determining the total variability of alkaline phosphatase. For serum albumin levels, however, these effects were determining a large portion of the total variability. The partial R^2 yielded an indication of the relative importance of the separate effects in the adjustment model. Thus the partial R^2 went beyond whether the adjustment for that effect was significant, and became a discriminating statistic for the relative importance of that adjustment. Coefficients of determination are summarized with least squares means and standard errors in Table III.

The effect of herd was significant for all traits in this

Table III. Least square means and associated standard errors (SE) with R^2 for all correction terms.

Trait	Mean	SE	R^2						
			Herd	Season	HxS	Age	Length	Stage	Total
Milk1	5994kg	45.2	.256*	.029*	.085*	.012*	.068*	na	.494
Fat 1	222.kg	1.79	.224*	.017*	.086*	.012*	.087*	na	.465
Prot1	195.kg	1.52	.262*	.020*	.064	.017*	.054*	na	.464
MilkC	7160kg	66.2	.184*	.009*	.050*	.108*	.127*	na	.553
Fat C	262.kg	2.59	.166*	.005*	.056*	.120*	.127*	na	.505
ProtC	230.kg	2.11	.198*	.005*	.053*	.095*	.142*	na	.533
Ca	9.54mg/dl	.021	.230*	.003	.206*	.008*	na	.002	.471
PO4	4.88mg/dl	.039	.114*	.000	.118*	.072*	na	.004	.354
Gluc	54.1mg/dl	.316	.188*	.036*	.298*	.002	na	.006*	.561
BUN	14.1mg/dl	.125	.333*	.001	.304*	.010*	na	.014*	.671
Uric	1.07mg/dl	.011	.165*	.104*	.119*	.000	na	.005*	.438
Chol	206.mg/dl	1.78	.210*	.018*	.121*	.003	na	.004	.375
T P	7.62g/dl	.022	.188*	.003	.086*	.154*	na	.003	.481
Alb	3.66g/dl	.013	.302*	.189*	.120*	.000	na	.001	.704
Bili	.170mg/dl	.004	.177*	.002	.063	.004	na	.010*	.317
Alk P	40.7mU/ml	.960	.089*	.001	.060	.038*	na	.002	.208
SGOT	136.mU/ml	1.18	.289*	.000	.133*	.002	na	.008*	.447
Creat	.957mg/dl	.005	.128*	.158*	.189*	.004*	na	.000	.552
Trig	9.68mg/dl	.121	.243*	.001	.136*	.009*	na	.002	.402
Na	141.meq/l	.127	.272*	.035*	.197*	.010*	na	.000	.542
K	4.24meq/l	.016	.142*	.001	.124*	.003	na	.000	.310
Cl	97.2meq/l	.108	.237*	.029*	.240*	.003	na	.006*	.570
HCO3	23.3meq/l	.080	.276*	.092*	.223*	.002	na	.001	.622
T4	5.25μg/dl	.058	.131*	.016*	.216*	.017*	na	.024*	.470
Amyl	193.Som.U	3.58	.159*	.008*	.073	.024*	na	.000	.306

* Significant at $P \leq 0.05$

study, accounting for between 8.9% and 33.3% of total variability. This reflected the effects of nutrition, management and microenvironment associated with each herd, and agreed with earlier papers (Hewett 1974, Payne et al. 1974, Rowlands and Manston 1976) which stated that herd was the single most important factor in determining the level of serum constituents.

The other factors in the model were not as universally significant or as important as the herd effect. They are discussed in detail at this point.

Season was significant for all milk production traits but accounted for only a small proportion of the variability. Adjustment for season has long been recognized by various dairy recording schemes, and this adjustment was warranted in view of the seasonal changes in many aspects of the cow's environment. Included in these seasonal changes were the change from pasture to stored feed, a management system still present on many farms. Other farms utilized stored feeds on a year-round basis. Also, since all herds did not experience a parallel seasonal change in feed, a significant interaction between herd and season was expected. This was found for all production traits with the exception of first lactation protein yield. It was noted that season was significant for all production traits but accounted for a much larger proportion of the variability in the current lactation traits. This indicated that, while the least squares constants were similar (Table II), there was relatively more

residual error involved in the current lactation traits. This resulted in less variability accounted for by season and thus a lower partial R^2.

Adjustment factors for age and lactation length have also been utilized as a means of standardizing production records. The rationale for this was reinforced by the significance of these two factors in determining the variability of all milk production traits.

A similar situation existed for age and length as did for seasonal effects. Regarding age, the least squares constants were higher for first lactation compared to current lactation traits, yet partial R^2 were higher for current lactation traits. This indicated that although the regression of first lactation on age exhibited a larger slope, the residual variability around this regression was large. This resulted in lower partial R^2, an indication of the lower overall strength of the regression. For length of lactation, least squares constants were similar for first and current lactation traits, yet residual variability, with respect to lactation length, was greater in first lactation. Again, this resulted in a lower partial R^2 for first lactation traits.

For the serum constituents, the significance and interpretation of the adjustment terms was not as straightforward as for the production traits. Although herd effects were significant and, in general, accounted for the

largest portion of the variability, the other adjustment factors varied greatly with regards to significance and relative importance.

Herd effect on levels of serum traits was a reflection of changes in the metabolism brought about by the differences in nutrition, feeding, management, and microenvironment among herds. It was expected that various metabolic activities in the animal will be altered due to differences in inputs to the animal and differences in the environment in which the animal functions. This alteration was expected to have its effect both directly on the metabolic pathway involving the particular serum constituents, or indirectly by affecting other constituents which are interrelated. Seasonal effects were expected to chronicle the seasonal changes in inputs and environment. Specifically, the change from pasture to stored feed altered greatly the nutritional inputs to the animal's metabolism and a quantitative change in some serum traits could be expected. As previously mentioned, if these seasonal changes were not parallel over all herds, a herd by season interaction could be expected.

The covariables associated with the serum traits, age at lactation start and stage of lactation at time of blood sampling, were both associated with changes in metabolism over time. As the animal matures, metabolic and hormonal changes occur and affect the levels of some serum constituents. As stage of lactation proceeded, hormonal and nutritional balance

stress were both invclved in the altering of some serum constituents.

Calcium and inorganic phosphate, which are interrelated in a number of body systems, showed the same trends for the adjustment factors. Herd, herd by season interaction and age effects were significant. This was in agreement with Tumbleson et al. (1973b) and Kitchenham et al. (1975) and (1976). Calcium and inorganic phospate have been reported (Ross and Halliday 1976) to be affected by season, but other reports (Payne et al. 1974) found no such significance. Stage of lactation has been variously reported to have a significant effect (Rowlands et al. 1975) or a nonsignificant effect (Hewett 1974) on calcium levels, so the fact that this study reported an insignificant lactational effect was not seriously disruptive.

Glucose was significantly affected by herd, season, herd by season interaction and stage of lactation. Seasonal influence on serum glucose has not been previously reported as significant (Payne et al. 1974). The significance of age effects on glucose have been in disagreement (Kitchenham et al. 1975, Kitchenham and Rowlands 1976 and Tumbleson and Hutcheson 1971) but was insignificant in this present study.

BUN was significantly affected by herd, interaction, age and stage of lactation. In view of the effect of diet on serum levels of BUN (Little and Manston 1972, Manston et al. 1975, and Prewitt et al. 1971), it was expected to be significant. However, as pointed out earlier, management on many farms now

includes stored feeds exclusively. This would lessen the within herd seasonal effect on any constituents largely affected by major changes in diet. This in turn indicated support for the significance of the interaction term.

Uric acid was significantly affected by all factors with the exception of age.

The serum lipids measured were cholesterol and triglycerides, although there was little similarity between these constituents for the significance of the adjustment terms. Cholesterol was adjusted for herd, season and interaction effects while triglycerides were adjusted for herd, interaction and age effects. The above situation for cholesterol was in general agreement with previously published studies with the exception that age was noted by Peterson and Waldern (1978) and by Tumbleson and Hutcheson (1971) to have a significant effect on cholesterol level. This age effect was dependent on the range of age present, which could explain the finding in this study.

Measures of protein in the serum were total protein and albumin. Albumin was significantly affected by herd, age and interaction, while total protein received further adjustment for age effects. This was in close agreement with Tumbleson *et al.* (1973b) who reported total protein but not albumin to be significantly affected by age. In early lactation, Rowlands *et al.* (1975) and Little (1974) found albumin and total protein both to be significantly affected by

stage of lactation, a result not forthcoming from the present study. Hewett (1974), using a range of lactation stage more closely approximating that in the present study, found no relationship of stage of lactation with albumin.

Bilirubin was affected by herd and stage of lactation. Tumbleson and Hutcheson (1971) and Mylrea and Healy (1968) differed with the present study in stating bilirubin was also affected by age.

Alkaline phosphatase was affected by herd and age. This was in agreement with previcus studies (Tumbleson et al. 1973a). SGOT was adjusted for herd, interaction and stage of lactation. This indicated that nutritional effects which comprised part of the herd and interaction terms were significant while climatic changes were not. The results for SGOT in the present study differed from past studies in that age has been cited as an effect (Peterson and Waldern 1978).

All factors except stage of lactation were utilized in adjusting creatinine levels. The available literature on this constituent supported this present finding (Peterson and Waldern 1978 and Mylrea and Healy 1968).

Sodium, potassium, chloride and bicarbonate are electrolytes in the blood which are important in many bodily functions and are instrumental in maintaining osmotic equilibrium. All four constituents were affected by herd, season and interaction factors with the exception of the seasonal effect on potassium. Additionally, sodium was

significantly affected by age and chloride by stage of lactation. The absence of age effects was generally supported by previous studies (Tumbleson et al. 1973b and Kitchenham et al. 1976).

Thyroxine was significantly affected by all factors. This agreed with results of Flamboe and Reineke (1959) and Mixner et al. (1962).

Amylase was significantly affected by herd, season and age.

Once significant systematic, environmental effects were eliminated from the data, it was presumed that only genetic and random environmental variability remained. Because of the design of this present study, the genetic and environmental components were separable with high relative efficiency. This separation yielded estimates of variance components, heritabilities, and genetic, environmental and phenotypic correlations. Since this present study estimated only the additive portion of the genetic variability and had no means of estimating nonadditive genetic variability, it was assumed in this study that the environmental parameters included all effects exclusive of the additive effect.

Table IV represents a summary of the genetic, environmental and phenotypic variances and the heritabilities associated with each trait. It was apparent that some genetic components of variance and some heritabilities were arbitrarily set to zero. This was instigated in cases where the genetic variance was

Table IV. Variance components and heritablities with associated SE for the individual traits.

Trait	k	V(A)±SE	V(E)±SE	V(P)±SE	h^2±SE
Milk1	15.25	68728±64289	571378±69953	640106±43127	.1074±.0976
Fat 1	15.25	153.9±113.9	852.2±116.2	1006.±68.37	.1530±.1078
Prot1	13.56	106.0±83.77	576.2±85.33	682.3±49.09	.1554±.1172
MilkC	17.35	459265±217238	903688±187688	1362593±93041	.3370±.1382
Fat C	17.35	587.5±297.6	1495.±265.9	2082.±138.8	.2821±.1270
ProtC	15.52	534.7±249.6	809.2±210.9	1344.±99.14	.3979±.1560
Ca	18.10	0.0	.1950±.0167	.1950±.1190	0.0
PO4	17.48	.0086±.0409	.6078±.0550	.6164±.0384	.0139±.0664
Gluc	18.13	0.0	42.78±3.661	42.78±2.618	0.0
BUN	18.14	.3057±.4792	6.312±.5952	6.618±.4060	.0462±.0718
Uric	18.17	.0002±.0034	.0548±.0047	.0550±.0034	.0045±.0616
Chol	18.06	0.0	1352.±116.1	1352.±82.92	0.0
T P	18.13	.0015±.0133	.2119±.0184	.2134±.0131	.0070±.0624
Alb	18.13	.0064±.0063	.0689±.0071	.0753±.0046	.0852±.0810
Bili	18.03	0.0	.0068±.0006	.0068±.0004	0.0
Alk P	18.06	116.4±56.74	272.4±49.94	388.8±25.65	.2994±.1283
SGOT	17.93	30.53±43.73	554.9±53.46	585.4±36.14	.0522±.0739
Creat	18.28	.0035±.0016	.0073±.0014	.0108±.0007	.3248±.1328
Trig	18.17	0.0	6.339±.5417	6.339±.3876	0.0
Na	17.83	0.0	6.950±.6025	6.950±.4290	0.0
K	17.83	.0144±.0108	.0972±.0113	.1116±.0070	.1288±.0921
Cl	17.87	.0837±.3218	4.812±.4310	4.896±.3020	.0171±.0657
HCO3	17.87	0.0	2.720±.2354	2.720±.1677	0.0
T4	18.44	0.0	1.472±.1244	1.472±.0893	0.0
Amyl	17.82	1082.±630.1	4265.±601.6	5347.±341.9	.2024±.1085

negative, resulting in a negative heritability. Since the theoretical lower limit of heritability is zero, a negative value was taken as a statistical estimate of a heritability that was very close to zero. Taken another way, a negative heritability was one that was not significantly different from zero.

Comparison of the components of variance estimates from this and previous studies was possible for the milk production traits. The present study used both first and current lactations. It was noted that current lactations were indeed first lactations if the animals were sampled during their first lactation. Any comparisons between estimates based on first or on current lactations were subject to considerations with regards to the degree to which the different lactation measures comply with the assumptions required by modern genetic theory. Historically, first lactation records have been considered more reliable insofar as they are less biased by selection or preferential feeding and management.

Estimated components of variance reported by Butcher et al. (1967) on all lactations agreed reasonably well with those estimated from current lactations in this present study. Variance components associated with first lactation were somewhat lower than these estimates. This discrepancy could be due to a number of factors. Since first lactation production was lower than current level production, it appeared that the

variance and the mean of these distributions were correlated, indicating a nonnormal distribution. Another possible cause could have been that older sires tended to have daughters in lactations subsequent to the first lactation, while younger sires would have daughters predominantly in their first lactations. This indicated that sire effect and age were confounded to some extent or that the age adjustment was somewhat inadequate for current lactation.

The heritabilities for the production traits fall into the range generally reported in the literature (Butcher et al. 1967, Johnson 1957). Heritability estimates for the current lactation production traits were considerably higher than those for first lactation. This may have arisen due to two aspects of the statistical analysis used in this study. As previously mentioned, sire effect and age were somewhat confounded in current lactation with the result that the additive variances and the heritabilities were inflated. Also, first lactation records were completed over a number of years preceeding the year of blood sampling. Since age, not year, was accounted for in the model, an inadequate adjustment may have occurred with a resulting inflation of environmental variance for first lactation. This would tend to reduce the heritability for first lactation traits.

In discussing the heritabilities of serum traits an important point must be considered. For production traits, the

adjustment model involved effects and covariables that were, with one minor exception, significant. For many serum traits, not all of the effects in the adjustment model were significant. This became important in that if certain terms were insignificant then the environmental variance would tend to be overestimated. This arose from two causes. Firstly, an insignificant adjustment term did not significantly reduce the error sums of squares. However, the insignificant term still removed degrees of freedom from the error line in the sire analysis. These had the combined effect of overestimating the environmental variance and thus underestimating the genetic variance. The wasted degrees of freedom became especially important when herd or interaction terms were insignificant, since 34 degrees of freedom were involved for both terms. If these terms were insignificant then the heritability estimated for that trait may have been underestimated.

Heritability estimates for serum constituents were in close agreement with estimates present in the literature. Albumin was significantly heritable in this study (h^2=0.085), an estimate in agreement of significance, if not magnitude, with Kitchenham and Rowlands (1976), Bettini *et al.* (1975) and Rowlands *et al.* (1974). The overall impression from the previous research was that the significant, low heritability estimated for albumin in this study was not unsupported in view of previous estimates and associated errors of estimation.

Alkaline phosphatase was estimated to have a moderate heritability (0.299). This estimate was higher than those previously reported but was in agreement of significance (Bettini et al. 1975, Peterson and Waldern 1978). It was further noted that the interaction adjustment term was insignificant, indicating that this estimate may be an underestimate.

The heritability estimate for creatinine was moderate to high (0.325). Peterson and Waldern (1978) reported a repeatability for this constituent of 0.514 indicating that the present estimate of heritability was in a corresponding range.

Potassium was heritable (0.129), a result in agreement with studies by Kitchenham and Rowlands (1976) and Rowlands et al. (1974). The magnitude of previous estimates were substantially higher than the present one but standard errors were also correspondingly higher indicating the lower relative reliability of the higher estimates.

Amylase, with a moderate heritability of 0.202, had no corroboration from previous studies. In view of its magnitude and small relative standard error, it is proposed that the present estimate was a reliable one. Given that the interaction adjustment was insignificant for amylase, this heritability may indeed be an underestimate.

Two constituents utilized in this study, BUN and SGOT, exhibited heritabilities below the critical significance level. These estimates were 0.046 and 0.052 for BUN and SGOT

respectively. Kitchenham and Rowlands (1976) reported a high heritability and Bettini et al. (1975) reported a low heritability for BUN, both estimates being significant. Rowlands et al. (1974) reported a non significant heritability for BUN, but this estimate was done on animals completely removed from the age range present in this study. SGOT has been reported as significantly heritable (Bettini et al. 1975) and as not significantly heritable (Furtmayr 1975). It was further noted that the standard errors of heritability for these two components allow a range of confidence within which a nonzero value for the population estimate will exist. Since, for these two constituents, this range of confidence reached into values that could prove effective in a selection program, BUN and SGOT may be interesting in spite of their insignificant heritabilities.

The remaining serum traits were not significantly heritable. This may have been due to two separate causes. One possibility was that there was no additive genetic variance present indicating fixation of the genotype responsible for the particular trait. The other possible cause could have been failure to fully identify all nongenetic sources of variation. These include environmental effects which were not random but were not able to be measured in the context of the present study. The second possible cause should be considered seriously for those traits of marginally significant heritability. In future studies, additional environmental effects should be

quantified to determine if traits found to be marginally heritable in this study have a significant genetic component.

Cholesterol was one serum constituent reported in some previous studies to have a large genetic component (Arave et al. 1975, Stufflebeam and Lasley 1969, and Taylor et al. 1966). Furtmayr (1975) did not concur with this finding. In the present study, heritability of cholesterol was close to zero and, considering the large relative standard error, the low estimate would not be in dispute.

Glucose was in a situation similar to that of cholesterol. Rowlands et al. (1974), Bettini et al. (1975) and Furtmayr (1976) indicated significant heritability while Kitchenham and Rowlands (1976) reported no such significnace. The estimate derived from the present study indicated the heritability to be negligible.

Calcium has been reported to be heritable by three previous studies (Kitchenham and Rowlands 1976, Rowlands et al. 1974 and Bettini et al. 1975). These reports indicated a low heritability for calcium and, in view of the standard errors involved, could not be considered secure. This present study was clear in its statement that calcium was negligibly heritable.

Inorganic phosphate was not heritable on the basis of this present study. This finding was both supported and refuted in the literature. Rowlands et al. (1974), in a study using calves, reported a low to moderate heritability for inorganic

phosphate, while Kitchenham and Rowlands (1976) and Wilson and Dinkel (1968) report no significance.

Total protein, shown significantly heritable by Kitchenham and Rowlands (1976), was not found to be so in the present study.

Uric acid was not found to be significantly heritable in this study. Bettini et al. (1975) reported a high heritability (0.47) but no indication of reliability was given. There was no further corroboration available.

The heritability of bilirubin was estimated as negative in this study. This was taken as an indication that heritability was negligible. Furtmayr (1975) reported bilirubin heritability as being insignificant. Bettini et al. (1975) reported a significant heritability but one which was low enough to be of little practical value.

Heritabilities of triglycerides, sodium, chloride, bicarbonate and thyroxine were all estimated as insignificant. No publications to the contrary have been located by this author.

A summary of phenotypic and environmental correlations among all traits is given in Table V. No discussion of the agreement between the results of this and previous studies will be given at this point. One reason is the large number of correlations to discuss. Another is that correlations reported in the literature are seldom of the same statistical derivation

Table V. Phenotypic (below diagonal) and environmental (above diagonal) correlations among traits with associated SE.

TRAIT	Milk1	Fat 1	Prot1	MilkC	Fat C	ProtC	Ca	PO4	Gluc
Milk1		.756±.049	.866±.024	.430±.092	.209±.100	.356±.105	.055±.083	-.113±.088	-.134±.082
Fat 1	.698±.039		.752±.047	.297±.120	.408±.094	.312±.124	.078±.086	-.190±.091	-.095±.088
Prot1	.876±.016	.755±.032		.350±.120	.208±.114	.391±.120	.059±.094	-.158±.096	-.079±.095
MilkC	.441±.061	.247±.078	.335±.077		.769±.050	.912±.023	-.000±.101	-.064±.096	-.108±.098
Fat C	.278±.067	.459±.060	.292±.073	.789±.029		.785±.045	.104±.092	-.081±.090	-.147±.087
ProtC	.409±.066	.341±.077	.428±.072	.923±.012	.832±.023		.045±.115	-.059±.102	-.071±.125
Ca	.050±.062	.069±.063	.052±.068	-.000±.066	.083±.063	.032±.071		.008±.065	.103±.062
PO4	-.097±.065	-.129±.066	-.138±.068	-.052±.066	-.074±.064	-.052±.069	.008±.052		-.105±.065
Gluc	-.122±.061	-.083±.064	-.069±.068	-.098±.066	-.113±.062	-.062±.075	.101±.049	-.102±.051	
BUN	.056±.065	.082±.066	.041±.075	.061±.068	.120±.065	.062±.077	.048±.052	.113±.054	-.042±.052
Uric	.083±.067	.037±.065	.042±.074	.030±.067	.049±.062	.095±.070	.169±.049	.184±.050	.109±.050
Chol	.016±.062	-.014±.064	-.012±.068	.275±.063	.220±.061	.247±.069	.133±.048	-.096±.051	-.007±.049
T P	-.049±.068	-.044±.070	-.051±.074	.015±.064	-.016±.062	.049±.068	.195±.048	-.056±.052	-.045±.050
Alb	.098±.065	.037±.069	.052±.076	.035±.072	.132±.066	.101±.078	.361±.047	-.073±.056	.033±.053
Bili	-.362±.054	-.042±.064	-.109±.067	.000±.066	.044±.064	-.019±.072	.016±.049	.063±.052	.301±.045
Alk P	-.049±.074	-.072±.076	-.006±.082	-.195±.080	-.244±.074	-.202±.086	.027±.062	.058±.064	.072±.062
SGOT	.019±.061	-.001±.064	.087±.066	.067±.070	.013±.068	.043±.073	-.048±.053	.078±.054	.003±.052
Creat	-.093±.074	-.112±.077	-.055±.085	-.023±.087	-.132±.081	-.119±.095	.006±.063	.102±.066	.030±.062
Trig	-.167±.060	-.133±.063	-.169±.067	-.102±.065	-.080±.063	-.116±.072	.283±.045	.048±.053	.147±.048
Na	-.039±.062	-.039±.064	.002±.069	.055±.065	.058±.062	.046±.071	.067±.049	.134±.052	-.076±.051
K	.118±.068	-.008±.071	.013±.076	.029±.074	.058±.069	.102±.078	.129±.056	.375±.051	-.032±.100
Cl	.008±.063	.010±.064	.043±.069	-.069±.067	-.063±.065	-.070±.072	.000±.052	-.199±.051	.100±.051
HCO3	-.093±.064	-.038±.066	-.056±.069	-.030±.066	-.016±.063	-.023±.072	-.047±.049	.212±.051	-.031±.051
T4	-.019±.061	.020±.063	.000±.068	-.041±.065	-.029±.063	-.099±.071	.120±.048	-.069±.051	.163±.048
Amyl	-.019±.074	-.043±.080	-.026±.083	.064±.075	.015±.073	.042±.079	.120±.058	-.091±.062	.154±.058

Table V(cont.). Phenotypic (below diagonal) and environmental (above diagonal) correlations among traits with associated SE.

TRAIT	BUN	Uric	Chol	T P	Alb	Bili	Alk P	SGOT	Creat
Milk1	-.029±.090	-.099±.095	.018±.084	.022±.095	.038±.090	-.395±.071	.124±.114	.028±.082	-.003±.112
Fat 1	.034±.093	-.049±.094	-.016±.089	.037±.100	-.022±.096	-.048±.088	.052±.117	-.002±.089	.031±.122
Prot1	-.078±.111	-.161±.110	-.014±.096	.059±.107	.088±.110	-.123±.093	.191±.128	.102±.092	.095±.140
MilkC	.056±.100	-.037±.104	.364±.096	-.019±.094	-.140±.115	-.000±.102	.116±.159	.067±.105	.107±.148
Fat C	.123±.092	.052±.090	.274±.088	-.019±.088	.039±.098	.055±.092	.011±.138	-.001±.099	.006±.136
ProtC	.041±.119	.101±.114	.340±.110	.053±.103	-.023±.126	-.027±.115	.121±.178	.076±.110	.175±.198
Ca	.050±.066	.173±.062	.134±.060	.199±.061	.386±.060	.016±.061	.034±.089	-.051±.067	.008±.093
PO4	.119±.069	.189±.063	-.099±.065	-.058±.065	-.080±.074	.065±.066	.061±.094	.086±.070	.122±.099
Gluc	-.044±.066	.111±.062	-.007±.062	-.045±.062	.035±.069	.303±.056	.091±.089	.003±.067	.038±.091
BUN		.187±.064	.189±.064	-.137±.065	-.011±.075	.200±.063	-.021±.091	.039±.070	-.051±.093
Uric	.184±.050		.031±.061	-.052±.063	-.076±.069	.432±.050	.012±.086	.009±.067	-.041±.090
Chol	.181±.050	.031±.049		.060±.063	.286±.065	-.098±.061	-.122±.089	.043±.067	-.002±.093
T P	-.130±.051	-.051±.050	.059±.050		.003±.070	-.179±.062	-.115±.086	-.057±.067	-.056±.090
Alb	.080±.056	-.069±.054	.265±.051	.003±.054		-.037±.070	-.064±.095	-.090±.076	.218±.103
Bili	.192±.050	.428±.040	-.097±.049	-.176±.049	-.034±.054		-.011±.089	.007±.067	.000±.093
Alk P	-.023±.064	.011±.061	-.097±.061	-.102±.061	-.064±.066	-.009±.062		.274±.104	.005±.127
SGOT	.037±.054	.008±.052	.041±.052	-.054±.053	-.020±.057	.007±.053	.087±.069		-.009±.098
Creat	-.066±.064	-.037±.061	-.001±.063	-.041±.061	.119±.070	.000±.063	.105±.079	.031±.067	
Trig	-.003±.053	.066±.051	.090±.049	.101±.050	.175±.053	.052±.049	.161±.061	.024±.053	-.441±.056
Na	.004±.053	.053±.051	.037±.050	-.127±.050	.157±.054	.065±.062	.004±.062	.120±.054	.119±.063
K	.122±.058	.170±.054	.033±.056	.068±.056	.059±.061	.034±.056	-.084±.068	.034±.060	.105±.073
Cl	-.092±.053	-.026±.052	-.075±.052	-.065±.051	.128±.054	.022±.052	.039±.062	.060±.054	.068±.061
HCO3	-.004±.053	.055±.050	-.010±.050	-.155±.049	-.130±.054	.079±.049	-.023±.062	-.004±.054	-.007±.063
T4	.012±.052	-.018±.050	.050±.049	-.054±.050	.258±.050	-.034±.049	.086±.061	.004±.052	.026±.062
Amyl	-.026±.062	.030±.061	.093±.058	.083±.059	.146±.063	.037±.059	-.009±.075	.029±.063	.063±.079

Table V(cont.). Phenotypic (below diagonal) and environmental (above diagonal) correlations among traits with associated SE.

TRAIT	Trig	Na	K	Cl	HCO3	T4	Amyl
Milk1	-.186±.081	-.043±.084	-.005±.099	.009±.085	-.139±.087	-.021±.082	.069±.108
Fat 1	-.152±.086	-.045±.089	.004±.101	.012±.089	-.047±.091	.023±.086	.143±.127
Prot1	-.196±.095	.002±.097	-.118±.112	.049±.097	-.064±.097	.000±.094	.129±.130
MilkC	-.137±.100	.071±.096	-.116±.114	.026±.101	-.040±.100	-.054±.097	-.013±.114
Fat C	-.101±.091	.070±.088	.043±.098	-.087±.096	-.020±.090	-.036±.090	.008±.106
ProtC	-.166±.118	.061±.111	.009±.123	.109±.118	-.031±.115	-.138±.114	.037±.123
Ca	.283±.056	.067±.062	.142±.073	-.000±.066	-.047±.062	.120±.060	.139±.079
PO4	.049±.067	.138±.066	.412±.066	-.206±.065	.219±.064	-.071±.065	-.108±.086
Gluc	.148±.060	-.078±.064	-.040±.051	.104±.065	-.032±.064	.164±.060	.178±.078
BUN	-.003±.068	.004±.068	.188±.077	-.096±.067	-.004±.068	.013±.066	.105±.087
Uric	.068±.064	.054±.064	.176±.070	-.026±.065	.055±.063	-.019±.063	.050±.084
Chol	.090±.061	.037±.062	.036±.074	-.077±.066	-.010±.062	.050±.061	.107±.078
T P	.104±.064	-.128±.062	.076±.074	-.066±.065	-.157±.062	-.055±.063	.088±.079
Alb	.190±.068	.170±.070	-.025±.083	.139±.070	-.140±.070	.277±.064	.169±.085
Bili	.052±.061	.065±.062	.037±.073	.022±.066	.079±.062	-.034±.061	.043±.080
Alk P	.203±.088	.004±.088	.036±.102	.041±.087	-.028±.088	.109±.089	.123±.115
SGOT	.025±.068	.128±.069	-.060±.080	.064±.070	-.004±.069	.004±.067	-.115±.088
Creat	-.576±.086	.155±.093	.245±.111	.074±.090	-.009±.093	.034±.092	-.135±.131
Trig		-.048±.062	.054±.074	-.138±.064	-.053±.062	.100±.060	.172±.084
Na	-.048±.049		.242±.071	.256±.061	.288±.056	.005±.061	-.042±.082
K	.049±.056	.217±.054		.050±.074	-.006±.074	-.057±.073	-.021±.093
Cl	-.134±.051	.249±.048	.048±.056		-.351±.057	.003±.065	.124±.082
HCO3	-.053±.049	.288±.045	-.005±.056	-.341±.045		-.042±.061	-.164±.081
T4	.100±.048	.005±.049	-.052±.056	.002±.051	-.042±.049		.-97±.080
Amyl	.142±.060	-.036±.060	.031±.066	.114±.059	-.140±.059	.083±.058	

nor were the data adjusted for the same effects as is done in the present study. A discussion of certain relationships will be undertaken in a later section.

A summary of genetic components of covariance and genetic correlation is given in Table VI. It is noted that many covariance components and many genetic correlations were set to zero. This was done for the same reason that heritabilities of some traits were set to zero. If the genetic component of variance for the individual trait was negative, this was considered as a statistical estimation of a parameter that has a lower limit of zero. The obtaining of a negative value was treated as a statistical indication that there was no genetic component of variance. The same rationale could be appropriate for some genetic components of variance that were only slightly greater than zero. If a negative or a very low genetic component of variance was used to estimate genetic correlations, the estimate of these genetic correlations were subject to extreme fluctuation, the exact value of which may greatly exceed the theoretical limits of the correlation statistic. Due to chance, correlations generated for these traits may fall in the acceptable range, but large standard errors were indicative of the lack of credibility attached to correlations between two traits with extremely low genetic components of variance.

On a theoretical basis it can be seen that if a trait has no genetic component, then it cannot exhibit a genetic

Table VI. Genetic correlations (below diagonal) and genetic components of covariance (above diagonal) with associated SE.

TRAIT	Milk1	Fat 1	Prot1	MilkC	Fat C	ProtC	Ca	PO4	Gluc
Milk1		1021±1822	2685±2710	9.67E[1]±6.80E	3866±2206	4026±2600	0.0	0.0	0.0
Fat 1	.314±.493		110.±98.5	947.±1110	199.±125.	134.±85.5	0.0	0.0	0.0
Prot1	.944±.101	.778±.215		2197±1762	153.±88.8	139.±85.2	0.0	0.0	0.0
MilkC	.588±.371	.107±.393	.302±.389		1.38E±.75E	1.46E±.82E	0.0	0.0	0.0
Fat C	.680±.430	.652±.283	.643±.371	.839±.101		516.±290.	0.0	0.0	0.0
ProtC	.701±.358	.437±.339	.557±.298	.949±.035	.966±.053		0.0	0.0	0.0
Ca	0.0	0.0	0.0	0.0	0.0	0.0		0.0	0.0
PO4	0.0	0.0	0.0	0.0	0.0	0.0	0.0		0.0
Gluc	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	
BUN	.944±.797	.494±.652	.768±.564	.134±.589	.158±.610	.158±.448	0.0	0.0	0.0
Uric	0.0	1.74±1.67	1.84±1.12	0.0	0.0	0.0	0.0	0.0	0.0
Chol	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
T P	-.597±.602	-.537±.516	-.799±.604	0.0	0.0	0.0	0.0	0.0	0.0
Alb	.637±.661	.456±.583	-.138±.549	.896±.513	.697±.484	.560±.393	0.0	0.0	0.0
Bili	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Alk P	-.964±.505	-.570±.396	-.906±.492	-.905±.190	-.911±.193	-.868±.203	0.0	0.0	0.0
SGOT	0.0	0.0	0.0	.100±.508	.107±.532	-.121±.619	0.0	0.0	0.0
Creat	-.490±.432	-.636±.357	-.546±.397	-.280±.297	-.448±.284	-.591±.244	0.0	.067±.712	0.0
Trig	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Na	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
K	.983±.530	-.078±.525	.836±.597	.616±.394	.137±.450	.444±.399	0.0	0.0	0.0
Cl	0.0	0.0	0.0	-.866±.990	0.0	0.0	0.0	0.0	0.0
HCO3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
T4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Amyl	-.495±.526	-.732±.368	-.653±.451	.328±.368	.039±.403	.069±.432	0.0	0.0	0.0

[1]E=x10⁴ for covariance and SE.

Table VI(cont.). Genetic correlations (below diagonal) and genetic components of covariance (above diagonal) with associated SE.

TRAIT	BUN	Uric	Chol	T P	Alb	Bili	Alk P	SGOT	Creat
Milk1	174.±73.7	0.0	0.0	-26.3±11.2	13.9±6.71	0.0	-2522±1013	0.0	-7.69±3.42
Fat 1	4.39±2.12	1.02±.428	0.0	-1.15±.479	.496±.209	0.0	-75.5±31.8	0.0	-.463±.199
Prot1	7.52±3.16	1.22±.503	0.0	-1.33±.569	-.158±.051	0.0	-92.7±37.1	0.0	-.354±.150
MilkC	52.4±30.8	0.0	0.0	0.0	47.0±18.8	0.0	-6153±2523	451.±290.	-11.7±4.47
Fat C	2.18±1.73	0.0	0.0	0.0	1.29±.589	0.0	-220.±93.5	17.3±7.31	-.662±.279
ProtC	3.04±1.56	0.0	0.0	0.0	1.23±.539	0.0	-207.±88.9	-15.3±4.69	-.890±.369
Ca	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
PO4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	.001±.001
Gluc	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
BUN		0.0	0.0	0.0	.064±.026	0.0	-.298±.178	0.0	-.006±.003
Uric	0.0		0.0	0.0	0.0	0.0	0.0	0.0	0.0
Chol	0.0	0.0		0.0	0.0	0.0	0.0	0.0	0.0
T P	0.0	0.0	0.0		0.0	0.0	0.0	0.0	0.0
Alb	1.45±1.09	0.0	0.0	0.0		0.0	-.072±.047	.418±.147	-.001±.000
Bili	0.0	0.0	0.0	0.0	0.0		0.0	0.0	0.0
Alk P	-.048±.585	0.0	0.0	0.0	-.084±.468	0.0		-65.3±22.1	.208±.087
SGOT	0.0	0.0	0.0	0.0	.919±.955	0.0	-1.01±.660		.095±.039
Creat	-.214±.598	0.0	0.0	0.0	-.260±.416	0.0	.345±.299	.266±.510	
Trig	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Na	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
K	-.701±.998	0.0	0.0	0.0	.757±.546	0.0	-.657±.394	1.10±.793	-.401±.400
Cl	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
HCO3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
T4	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Amyl	-1.29±.944	0.0	0.0	0.0	.008±.516	0.0	-.446±.346	1.46±1.03	.594±.283

Table VI(cont.). Genetic correlations (below diagonal) and genetic components of covariance (above diagonal) with associated SE.

TRAIT	Trig	Na	K	Cl	HCO3	T4	Amyl
Milk1	0.0	0.0	34.0±15.0	0.0	0.0	0.0	-4426±1770
Fat 1	0.0	0.0	-.130±.055	0.0	0.0	0.0	-354.±142.
Prot1	0.0	0.0	1.07±.436	0.0	0.0	0.0	-252.±104.
MilkC	0.0	0.0	47.8±18.5	-234.±96.6	0.0	0.0	6438±2710
Fat C	0.0	0.0	.367±.191	0.0	0.0	0.0	27.8±13.4
ProtC	0.0	0.0	1.24±.565	0.0	0.0	0.0	41.9±24.5
Ca	0.0	0.0	0.0	0.0	0.0	0.0	0.0
PO4	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gluc	0.0	0.0	0.0	0.0	0.0	0.0	0.0
BUN	0.0	0.0	-.043±.012	0.0	0.0	0.0	-22.6±8.60
Uric	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Chol	0.0	0.0	0.0	0.0	0.0	0.0	0.0
T P	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Alb	0.0	0.0	.007±.003	0.0	0.0	0.0	.023±.225
Bili	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Alk P	0.0	0.0	-.715±.292	0.0	0.0	0.0	-150.±56.4
SGOT	0.0	0.0	.723±.281	0.0	0.0	0.0	234.±89.5
Creat	0.0	0.0	-.002±.001	0.0	0.0	0.0	1.20±.466
Trig		0.0	0.0	0.0	0.0	0.0	0.0
Na	0.0		0.0	0.0	0.0	0.0	0.0
K	0.0	0.0		0.0	0.0	0.0	1.21±.487
Cl	0.0	0.0	0.0		0.0	0.0	0.0
HCO3	0.0	0.0	0.0	0.0		0.0	0.0
T4	0.0	0.0	0.0	0.0	0.0		0.0
Amyl	0.0	0.0	.317±.453	0.0	0.0	0.0	

correlation with another trait, regardless of whether the second trait has a genetic component. For this reason, when the above situation occurred or was indicated by extreme correlations and standard errors, the genetic component of covariance and the genetic correlation were set to zero. The appendix lists the number of paired observations used for each correlation.

Genetic improvement in dairy cattle has proceeded primarily by selection for the additive portion of the total gene effect, and it is unlikely that the dairy farmer will find a better breeding program within the range of conventional selection theory. To work within the framework of direct selection, the traits must have a significant genetic component. In general, a significant genetic component indicates that selection on that trait can result in movement of the population mean in the desired direction. In the context of this present study, it also indicated that a portion of the genotype of the cow was responsible for establishing the measured level of that trait, and that the additive effect of the genes were a major controlling factor in this genotype.

Also, with regards to conventional selection theory, another approach is that of indirect selection. This approach is based on the premise that the traits involved have a significant genetic correlation and thus selection for one trait will result in a concurrent change in another trait. The classical interpretation of this situation is that the

individual genotypes involved with the traits have some portion in common, the relative size of this portion being estimated by the genetic correlation. This common genotype affects the two traits through the phenomenon of pleiotropy of gene effect.

The idea of genetic correlation is that there must be significant genetic components for both traits. If a trait were shown to have no genetic component, then it follows that it cannot have any significant genetic correlations. On this basis, the number of traits meriting further discussion is reduced to those with a significant genetic component. These are creatinine, alkaline phosphatase, amylase, potassium, albumin, SGOT, BUN and all milk production traits. In further discussion of the various correlations only these traits will generally be considered.

Beyond the estimation of heritability of the serum traits, this study also examined some relationships involving these traits and the milk production traits. A summary of phenotypic and genetic correlations between the production traits and the heritable serum constituents is given in Table VII.

The genetic correlations were of greatest interest in this study but a word of caution in interpreting these correlations is warranted. The genetic correlations were calculated using halfsib analyses and multiplication by a constant of four was involved in the calculation. Since errors of measurement were also multiplied by this factor, the standard error associated

Table VII. Genetic (above) and phenotypic (below) correlations between selected serum constituents and the production traits.

Trait	h^2	Creat	Alk P	Amyl	K	Alb	SGOT	BUN
Milk1	.1074±.0976	-.490±.432	-.964±.505	-.495±.526	.983±.530	.637±.661	0.0	.944±.797
		-.093±.074	-.049±.074	-.019±.074	.118±.068	.098±.065	.019±.061	.056±.065
Fat 1	.1530±.1078	-.636±.357	-.570±.396	-.732±.368	-.078±.525	.456±.583	0.0	.494±.652
		-.112±.077	-.072±.076	-.043±.080	-.008±.071	.037±.069	-.001±.064	.082±.066
Prot1	.1554±.1172	-.546±.397	-.906±.492	-.653±.451	.836±.597	-.138±.549	0.0	.768±.564
		-.055±.085	-.006±.082	-.026±.083	.013±.076	.052±.076	.087±.066	.041±.075
MilkC	.3370±.1382	-.280±.297	-.905±.190	.328±.368	.616±.394	.896±.513	.100±.508	.134±.589
		-.023±.087	-.195±.080	.064±.075	.029±.074	.035±.072	.067±.070	.061±.068
Fat C	.2821±.1270	-.448±.284	-.911±.193	.039±.403	.137±.450	.697±.484	.107±.532	.158±.610
		-.132±.081	-.244±.074	.015±.073	.058±.069	.132±.066	.013±.068	.120±.065
ProtC	.3979±.1560	-.591±.244	-.868±.203	.069±.432	.444±.399	.560±.393	-.121±.619	.158±.448
		-.119±.095	-.202±.086	.042±.079	.102±.078	.101±.078	.043±.073	.062±.077

with the genetic correlation was appropriately inflated. The standard errors must be large for these correlations since they were estimated from several statistics, each one subject to its own sampling variance. The magnitude of the correlation is best judged in relation to its standard error.

First lactation milk production possessed strong genetic correlations with creatinine (-0.490), alkaline phosphatase (-0.964), potassium (0.983), albumin (0.637) and BUN (0.944). The reliability of these correlations was reflected in their standard errors. The strongest genetic correlation was between milk and alkaline phosphatase, with the standard error indicating a reliable estimate. The poorest correlation among the set of constituents listed above was for albumin. Although the correlation between creatinine and milk was less than that for albumin, the standard error was much lower, indicating a more reliable estimate.

Current lactation milk was correlated with a smaller subset of serum constituents. Alkaline phosphatase, as with first lactation milk, showed the strongest relationship (r=-0.905) with a small standard error. Potassium (0.616) and albumin (0.896) also had strong, reliable correlations with current lactation milk. Unlike first lactation milk, there was no strong relationship with creatinine or BUN. These discrepancies between what are essentially estimates of the same correlation taken at different points in time were not serious. The correlation estimates for creatinine and BUN were associated

with large standard errors. The fact that they appeared significant only for the first lactation may have indicated that the correlation importance was marginal and the two estimates of the correlation were different merely by chance.

Literature was sparse in reporting of relationships of serum constituents with milk production. Kitchenham et al. (1975) and Payne et al. (1973) reported a relationship between milk production and albumin, in agreement with this present study. Furtmayr (1975) reported a positive correlation with SGOT, a finding not supported by this present study. Other correlations between serum constituents and milk production estimated in this study were not discussed or were noted to be insignificant in the literature.

First lactation fat production was genetically correlated with creatinine (-0.636), alkaline phosphatase (-0.570) and amylase (-0.732). Current fat production followed a similar pattern except that amylase was not strongly correlated. Also, albumin appeared as highly correlated (0.697) for current fat only.

First lactation protein yield was correlated with creatinine (-0.546), alkaline phosphatase (-0.906), amylase (-0.653), potassium (0.836) and BUN (0.768). Current production was correlated with a smaller group of constituents. This group consisted of creatinine (-0.591), alkaline phosphatase (-0.868),

potassium (0.444) and albumin (0.560).

There is some discussion warranted on the relative merits of the two groups of correlations, those involving first lactation production and those involving current lactation. Current lactation records represented estimates that were indicative of the population as it existed at the time of bleeding. This association in time between current lactation traits and serum traits was important in that these relationships were reflections of current physiological events. Thus, if current feeding programs were a cause of a serum and production change, correlations will tend to be overestimated. This overestimation, however, should be present only in environmental and phenotypic correlations. Genetic correlations would be expected to be unaffected by such environmentally caused relationships. Beyond this, the comparison of first lactation versus current lactation correlations were subject to the same considerations as were discussed with respect to heritabilities. On balance, then, first lactation correlations with serum traits may be more reliable indicators of underlying genetic relationships for the following reasons. First lactation correlation estimates were conservative since environmental covariance was possibly overestimated due to the incomplete adjustment for year of calving. Secondly, current lactation correlations may have been inflated due to confounded age and sire effects. Both sets of correlation estimates were

used in the assessment of the genetic relationships between production and serum traits, with the reservation that those involving first lactation production were probably more realistic.

Furthermore, no clearcut distinctions were discernable between the two production groups on the basis of the estimates obtained in this study. The trends of the correlations already discussed were similar for both production trait groups. Some discrepancies arose but none were so serious or patterned as to be useful in declaring either first or current lactations as a preferred standard.

A more general picture of the relationships involving the production traits and the selected serum constituents may be discerned by discussing the serum traits that were most heavily involved. Creatinine and alkaline phosphatase, the serum traits with the highest heritability, were the traits most highly correlated with the production traits. These two constituents correlated highly negatively with all production traits except creatinine with current lactation milk. There was a particularly strong relationship between alkaline phosphatase and the current production traits. These relationships were shown to be reliable by the relatively low standard errors.

Potassium was another trait that was involved with milk production and retained a pattern for both production estimates. It correlated positively with milk and protein production but

did not show a strong relationship with milk fat.

The remaining selected serum constituents did not show strong, consistent correlations with the production constituents. Albumin was positively correlated with both measures of milk production but, with regards to fat and protein, the correlations were not significant for both estimates and thus were considered as not particularily strong. Amylase and BUN showed strong correlations with some milk traits but only in the first lactation. SGOT showed no strong correlations.

This study has focused on those traits which were judged to have a significant genetic component. Animal breeding theory states that progress due to selection is proportional to the genetic variance and the heritability of the trait. A small genetic component relative to the phenotypic variability will result in both parameter estimates being low. Thus, any traits with a low heritability were not deemed as important as those that posessed a significant additive component since the traits with low heritabilities would not be expected to progress under selection at an acceptable rate. In future studies, if one were able to more precisely identify this environmental component, the relative importance of the additive variance would increase. This would result in a more accurate assessment of the usefulness of uric acid and its genetic relationships with other traits.

Indirect selection offers an alternative to the direct selection of a trait in modern animal breeding theory. Response to direct selection is proportional to additive variance, heritability and selection differential. Response to indirect selection depends on the heritability of the trait used indirectly, the genetic variance of the trait in which change is sought, the genetic correlation between the two traits and the selection differential. The success of indirect selection compared to direct selection will depend on the relative magnitude of these factors. In practice, ease and economy of measurement may become a consideration in trait selection. A rational decision must be reached between the strength of the genetic system and the considerations of its practical use. Some illumination of the relationships between the serum traits will provide support for making this decision.

Table VIII presents the genetic and phenotypic correlations among those serum constituents judged to be significantly heritable. Included in this table are correlations of sufficient magnitude to be of interest and which have associated standard errors of low relative magnitude, indicating some degree of reliability. Unlike the correlations involving the milk production traits, there were not two estimates of the same correlation available. Therefore, there could be no corroboration within this present study of the estimates obtained.

Creatinine, the most heritable of the serum traits,

Table VIII. Genetic (above diagonal) and phenotypic (below diagonal) correlations among selected serum constituents.

Trait	h^2	Creat	Alk P	Amyl	K	Alb	SGOT	BUN
Creat	.3248±.1328		.345±.299	.594±.283	-.401±.400	-.260±.416	.266±.510	-.214±.598
Alk P	.2994±.1283	.105±.079		-.446±.346	-.657±.394	-.084±.468	-1.01±.660	-.048±.585
Amyl	.2024±.1085	.063±.079	-.009±.075		.317±.453	.008±.516	1.46±1.03	-1.29±.944
K	.1288±.0921	.105±.073	-.084±.068	.031±.066		.757±.546	1.10±.793	-.701±.998
Alb	.0852±.0810	.119±.070	-.064±.066	.146±.063	.059±.061		.919±.955	1.45±1.09
SGOT	.0522±.0739	.031±.067	.087±.069	.029±.063	.034±.060	-.020±.057		0.0
BUN	.0462±.0718	-.066±.064	-.023±.064	-.026±.062	.122±.058	.080±.056	.037±.054	

exhibited few strong genetic correlations with other serum constituents. With regards to genetic correlations with the heritable blood constituents, creatinine was related positively with alkaline phosphatase (0.345), although the standard error did not indicate that this degree of correlation was well defined. The correlation of creatinine with amylase was stronger (0.594) and more reliable as indicated by the standard error. It appeared that amylase and alkaline phosphatase have a genotype that is in part common with that of creatinine. Amylase was moderately heritable and the most efficient method of selection would be that of direct selection for amylase level.

SGOT, a trait which had a heritability judged to be statistically insignificant, had a number of strong correlations with the heritable serum constituents. It was previously pointed out that little progress could be expected under direct selection for SGOT since its heritability was low (0.052). Indirect selection would be an improvement if traits could be located with larger heritabilities and strong correlations with SGOT. Several serum traits possessed these characteristics. Amylase was moderately heritable (0.202) and had a correlation with SGOT of 1.46. This large correlation was reasonably reliable when judged on the basis of its standard error. This indicated a high degree of similarity in the genotypes responsible for the two traits. Correlations were only slightly less strong for SGOT with alkaline phosphatase (-1.01),

potassium (1.10) and albumin (0.919). A reasonable degree of success could be expected in moving the level of SGOT by selecting for any of these constituents.

BUN was in much the same situation as SGOT. Not significantly heritable, it possessed large genetic correlations with certain heritable traits. One of these traits was albumin, which has low, significant heritability and a genetic correlation of 1.45 with BUN. Amylase, which had a moderate heritability, had a correlation with BUN of -1.29. It was apparent that, on the basis of this study, the most successful method of altering the population level of BUN would be to select for a lower level of amylase.

It should be noted that some of these correlations involving SGOT and BUN exceeded the theoretical range of the correlation statistic to some degree. However, the standard errors were small enough, relative to the correlation estimates to indicate a degree of confidence in the high values for some correlations. That they exceeded the theoretical limit was a reflection of several factors. One was the fact that one of the traits involved had a small genetic component. Combined with rounding errors and errors involved in the estimation of variance and covariance components, this small genetic component caused wide fluctuations in the estimates. Due to the smaller standard errors, these correlations were taken to indicate that the genotypes of the traits involved were highly similar.

Other genetic correlations existed among serum constituents

and seemed reliable on the basis of their standard errors, but were not as large in magnitude as those already mentioned. Alkaline phosphatase was negatively correlated with amylase (-0.446) and this estimate was relatively reliable. Alkaline phosphatase also correlated negatively with potassium (-0.657) with a reasonable standard error. Finally, potassium correlated positively with albumin (0.757). These last three relationships were of limited usefulness. All three relationships involved heritable traits and therefore direct selection should be successful in altering population levels. The magnitude of the correlations among these three constituents was not sufficiently large to propose the use of indirect selection.

The preceeding discussion of the correlations estimated in this study pointed out the complex genetic relationships underlying some of the compounds and elements found in the serum of dairy cattle. Besides being of interest to geneticists, these relationships could prove interesting as background relationships in the interpretation of results from physiological experiments. As an example, SGOT and potassium exhibited a higher positive genetic correlation (1.100) but small environmental (-0.060) and phenotypic (0.034) correlations. This indicated a very similar genotype but a large environmental input on these traits. If a correlated response in these two traits were noted in a trial involving small numbers of animals, it could be due merely to a sampling anomaly rather than due to the treatments present in the trial.

The same rationale can be extended to all other correlations estimated in this study. A preliminary investigation of the results of this study would benefit researchers in other fields by forewarning them of the genetic consequences relevant to their present experiments.

CONCLUSION

This study has estimated and evaluated for some serum and milk production traits the genetic parameters that are necessary for using these traits in a breeding program. Among the parameters estimated were the genetic components of variance and the heritabilities of the individual traits. It was suggested that to be of greatest interest, a trait should possess moderate to high heritability. This would allow the animal breeder to change the level of expression of a trait if such a change were shown to be desirable. The desirability of a trait in this study was further evaluated in terms of the genetic relationships among the serum and production traits. Since it is probable that these represented only a small portion of the relationships of interest involving serum constituents in dairy cattle, this study was in part a report of genetic parameters of serum constituents as a reference for later studies.

The production of milk, milk fat and milk protein exhibited moderate heritability, a finding that was not unexpected. The serum traits that exhibited this degree of heritability were creatinine, alkaline phosphatase, amylase, potassium and albumin. SGOT and BUN exhibited low heritability but were hypothesized to be of possible importance on the basis of findings reported in the literature. This indicated that progress could be achieved by direct selection on the level of these serum constituents. The heritabilities estimated in this

study were reported as reference values for future studies involving the additive genetic components of these traits.

As an evaluation of the relationships between the serum traits and the production traits, genetic correlations were estimated and discussed particularly with regards to the magnitude of the associated standard errors. For the estimates of genetic correlation involving the milk production traits, two estimates were available in this study, one for current lactation and one for first lactation. First lactation estimates were considered somewhat superior to current lactation estimates. Both were used as separate estimates of the same parameter in the discussion. This in turn allowed the discerning of those relationships between serum traits and production traits that were of greatest reliablity.

The most consistent and reliable genetic correlations between serum and production traits were as follows. Serum level of potassium correlated highly and positively with milk production for both first and current lactations. Albumin level also possessed a high, positive correlation with milk production.

The strongest relationships involved alkaline phosphatase and creatinine with the production traits. Genetic correlations of alkaline phosphatase with all three production traits were negative and involved the highest magnitude of any of the correlations of the heritable serum traits with the production traits. Creatinine was negatively correlated with milk protein

and milk fat. Correlation of creatinine level with milk production was not strong.

These findings effectively reduced the original nineteen serum constituents into a subset that has shown a genetic component and/or has exhibited relationships with the production traits. This study has identified such a subset and estimated heritabilities and genetic correlations as indications of existing relationships, for the benefit of future studies.

Other relationships examined were those that existed among the serum constituents. This was investigated for two reasons. One was to assess the posibility of utilizing indirect selection in changing the level of a correlated, nonheritable trait. Second, and most important was to use the genetic correlation as an assessment of the underlying genotypic relationships. These genotypic relations could be of use to researchers in other fields in interpreting their results. Genetic correlations were again taken as the method of assessment. SGOT exhibited several strong genetic correlations - positive with amylase, potassium and albumin, and negative with alkaline phosphatase. Since alkaline phosphatase and amylase possessed the highest heritabilities of these traits, selection on these two traits should be successful in altering the serum level of SGOT, a trait of low heritability. BUN exhibited a strong negative correlation with amylase and a strong positive correlation with albumin. Since BUN had low heritability, selection on amylase level should result in an indirect change in BUN level.

Genetic correlations among the heritable serum traits were not as specifically useful in indirect selection but were indicative of underlying genetic relationships. Creatinine exhibited moderate positive correlations with alkaline phosphatase and amylase. Additionally, alkaline phosphatase exhibited moderate negative correlation with amylase and potassium. Finally, a moderate, positive correlation existed between potassium and albumin.

This study has been successful in identifying a subset of the serum traits measured that were of interest on the basis of the heritability of the trait and its genetic correlations with other serum and production traits. These genetic parameters are tabulated as a reference source as well as a source of guidance in future studies involving these traits.

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Appendix. Number of observations for each pair of traits.

TRAIT	Milk1	Fat 1	Prot1	MilkC	Fat C	ProtC	Ca	PO4	Gluc
Milk1									
Fat 1	450								
Prot1	401	401							
MilkC	426	426	382						
Fat C	426	426	382	512					
ProtC	388	388	382	459	459				
Ca	439	439	395	501	501	452			
PO4	424	424	386	487	487	441	514		
Gluc	440	440	395	502	502	452	533	516	
BUN	440	440	395	502	502	452	533	515	534
Uric	441	441	396	503	503	453	534	516	535
Chol	438	438	394	500	500	451	531	513	532
T P	440	440	396	502	502	453	533	515	534
Alb	440	440	395	502	502	452	533	516	535
Bili	437	437	393	499	499	450	530	513	531
Alk P	439	439	394	500	500	450	531	513	532
SGOT	434	434	391	496	496	448	527	511	528
Creat	445	445	398	506	506	454	529	511	530
Trig	444	444	396	504	504	452	526	508	527
Na	436	436	388	494	494	441	516	498	517
K	436	436	389	494	494	442	516	498	517
Cl	435	435	387	495	495	442	517	499	518
HCO3	435	435	387	495	495	442	517	499	518
T4	449	449	401	511	511	458	534	516	535
Amyl	435	435	388	494	494	442	516	499	518

Appendix(cont.). Number of observations for each pair of traits.

TRAIT	BUN	Uric	Chol	T P	Alb	Bili	Alk P	SGOT	Creat
Milk1									
Fat 1									
Prot1									
MilkC									
Fat C									
ProtC									
Ca									
PO4									
Gluc									
BUN									
Uric	535								
Chol	532	533							
T P	534	535	532						
Alb	534	535	532	534					
Bili	531	532	529	531	531				
Alk P	532	533	530	532	532	529			
SGOT	528	529	526	528	528	527	526		
Creat	530	531	528	530	530	527	528	524	
Trig	527	528	525	527	527	524	525	521	535
Na	517	518	515	517	517	514	515	511	523
K	517	518	515	517	517	514	515	511	523
Cl	518	519	516	518	518	515	516	512	524
HCO3	518	519	516	518	518	515	516	512	524
T4	535	536	533	535	535	532	533	529	539
Amyl	517	518	515	517	518	514	515	511	524

Appendix (cont.). Number of observations for each pair of traits.

TRAIT	Trig	Na	K	Cl	HCO3	T4	Amyl
Milk1							
Fat 1							
Prot1							
MilkC							
Fat C							
ProtC							
Ca							
PO4							
Gluc							
BUN							
Uric							
Chol							
T P							
Alb							
Bili							
Alk P							
SGOT							
Creat							
Trig							
Na	520						
K	520	525					
Cl	521	524	524				
HCO3	521	524	524	527			
T4	536	526	526	527	527		
Amyl	521	515	515	514	514	526	