CONCENTRATION AND DISTRIBUTION OF SIALIC ACID IN SOW MILK DURING LACTATION

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Background/Aims: Human milk sialic acids has been proposed as a bioactive compound promoting immune function, gut maturation and neurodevelopment of the newborn. Porcine milk however, has received little attention. The aims of the present study were to quantify and compare the levels of *N*-acetylneuraminic acid (Neu5Ac), *N*-glycolylneuraminic acid (Neu5Gc) and ketodeoxynonulpsonic acid (KDN) in sow milk during course of lactation.
Method: Milk samples from 22 sows were collected by manual expression on 3 occasions, day 1 (colostrum), day 3 (transition milk) and day 15-21 (mature milk) respectively. The concent-ration of Neu5Ac, Neu5Gc and KDN were analyzed using UHPLC.
Results: Sow milk contained significant amounts of Sia with the highest concentration found in colostrum (1238.50 mg/L) followed by transition milk and then mature milk. Neu5Ac was the major form of Sia (93-96%) and then Neu5Gc (3-6%), KDN however contained as little as 1-2%. This distribution was common to each milk fraction and to each time point in lactation.
Conclusions: Porcine milk contained a rich source of sialylated glycoconjugate. The predominately form of sialic acid is Neu5Ac. The high concentrations of Sia in porcine milk suggest that Sia is an important nutrient that may contribute to the optimization of immune function, neuro-development and growth and development of piglets.
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MOLECULAR MECHANISMS DRIVING AIRWAY INFLAMMATION FOLLOWING A HIGH-FAT MIXED MEAL IN ASTHMA

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Background/Aims: A high fat meal is associated with airway inflammation in asthma, but the mechanisms are not well understood. This aim of this study was to use microarray techniques to examine the molecular mechanisms of fat-induced airway inflammation in asthma compared with healthy controls.
Methods: Subjects with asthma ($n = 11$) and healthy controls ($n = 8$) were provided with a high-fat, high energy meal, containing total energy (TE) of 3846 kJ and 48 g (49% of TE) total fat, including 20.5 g (21% of TE) saturated fat. Sputum was induced at 0 and 4 hours and gene expression was examined by microarray and quantitative real-time PCR (qPCR).
Results: Following the high fat dietary challenge, 168 entities were significantly differentially expressed greater than 1.5 fold in subjects with asthma. Five genes involved in immune system processes were selected for qPCR analysis (*S100P, S100A16*, *MAL*, *MUC1* and *NLPR12*). qPCR confirmed that *S100P, S100A16*, *MAL* and *MUC1* were significantly increased in the asthma group post-meal. There was a moderate and significant correlation between the change in *S100P* and *MUC1* gene expression and the change in sputum %neutrophils following the high fat meal ($r = 0.552$, $p = 0.024$; $r = 0.495$, $p = 0.045$ respectively). *NLRP12* gene expression at 4 hours strongly correlated with the change in total and saturated non-esterified plasma fatty acid levels at 2 hours ($r = 0.555$, $p = 0.028$; $r = 0.53$, $p = 0.037$ respectively).
Conclusions: Our data identifies several genes that contribute to neutrophilic airway inflammation following consumption of a high fat meal in asthmatics, which may prove to be therapeutic targets for immunomodulation.
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DOES INCREASED INTAKE OF FOLIC ACID INCREASE CANCER RISK?

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Background/Aims: Food Standards Australia New Zealand monitored the literature regarding cancer risk following mandatory fortification with folic acid to reduce the risk of neural tube defects in Australia.
Methods: Randomised controlled trials testing folic acid for at least one year and reporting any of: all-cause mortality, total cancer, four selected cancers or recurrence of colorectal adenoma in healthy non-pregnant adults were identified by searching in PubMed and CENTRAL from 2001 onwards. Random effects meta-analysis of the relative risks was conducted using StatsDirect (http://www.statsdirect.co.uk).
Results: Twenty-six trials, lasting up to 7.3 years, were identified from 4216 abstracts. The larger trials generally described masked allocation and blind outcome assessment. The 13 trials (43,557 subjects) reporting total cancer incidence yielded a non-significant overall relative risk (RR) of 1.04 (95%CI: 0.97-1.11). Fewer studies reported results for colorectal cancer RR = 1.00 (95%CI: 0.82-1.23), breast cancer RR = 0.82 (95%CI: 0.63-1.07), lung cancer RR = 1.00 (95%CI: 0.84-1.21) or prostate cancer RR = 1.16 (95% CI: 0.85-1.60). Only the results for prostate cancer indicated any heterogeneity ($I^2 = 52.7\%$). Most data comes from large trials testing 0.8-2.5 mg/day.
Conclusions: There are no significant increases or decreases in cancer risk. Other meta-analyses using different inclusion criteria to select studies have found similar results. The amount of folic acid used in mandatory fortification in Australia is about one-tenth of that tested and the effect on blood folate levels has been commensurately lower.
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SAFETY OF EMU OIL FOR INTESTINAL APPLICATIONS

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Background/Aims: Previously, we have reported that Emu Oil (EO) lengthens intestinal crypts, a feature of intestinal growth, in experimental colitis (2012) and mucositis (2013). However, it remains unclear whether the crypt lengthening represents normal or aberrant intestinal growth. We aimed to determine if crypt depth measurements in EO-treated rats returned to normal levels following withdrawal of EO therapy.
Methods: Dark Agouti rats ($n = 8$/group) were gavaged daily for 10 days with Water, Olive Oil (OO) or EO (1ml) or 0.5 mL EO (0.5EO). Rats were euthanized on day 10 or day 17. Intestinal weights, lengths, villus height (VH) and crypt depth (CD) were quantified. $P < 0.05$ was considered significant.
Results: On day 10, jejuno-ileum (JI) weight was increased by OO (26%) and EO (0.5 mL: 15%; 1 mL: 29%; $p < 0.01$), which was normalised by day 17. On days 10 and 17, JI length was greater in OO- (12%) and EO-treated rats (0.5 mL: 8%; 1 mL: 12%; $p < 0.05$), relative to water controls. On day 10, OO and EO increased ileal VH (OO: 32%; 0.5EO: 22%; EO: 35%; $p < 0.01$) and CD (OO: 17%; 0.5EO: 13%; EO: 22%); importantly however, after withdrawal of all oils, VH and CD returned to normal levels. Moreover, the VH:CD ratio (a feature of dysplasia) was unaffected in all oil-treated rats compared to normal controls.
Conclusions: The restoration of normal intestinal growth following cessation of Emu Oil therapy supports its safety for intestinal conditions.
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DIETARY FISH OIL AT LOW INTAKES INCREASES DHA INCORPORATION AND REDUCES LOW FREQUENCY FATIGUE IN RAT HINDLIMB SKELETAL MUSCLE