

Sex and Sport-Specific Energy Demands Influence Lipid and Nitrogen Metabolite Profiles in Athletes

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Abstract

Cardiometabolic laboratory values in athletes may reflect sex, habitual training, sport energy demands, diet, hydration, and recent exercise. This study examined lipid and nitrogen-metabolite profiles across aerobic, anaerobic, and mixed-sport athletes and tested whether group patterns differed by sex. Summary data were available for 429 athletes (246 males and 183 females): aerobic (130 males, 66 females), anaerobic (25 males, 10 females), and mixed-sport (91 males, 107 females). Total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), very-low-density lipoprotein cholesterol, low-density lipoprotein cholesterol, total cholesterol/HDL-C ratio, urea, and uric acid were evaluated. Two-factor analyses reconstructed from cell means and standard deviations examined sex, sport category, and interaction effects. Sport category influenced every outcome (all $p < .001$; partial eta squared = .045-.066). Females had higher HDL-C and lower total cholesterol/HDL-C ratios, LDL-C, triglycerides, very-low-density lipoprotein cholesterol, and uric acid than males. Significant sex-by-sport interactions occurred for triglycerides and very-low-density lipoprotein cholesterol (both $p = .031$). Aerobic groups generally showed the most favourable lipid pattern but higher urea and uric acid, whereas anaerobic athletes had the highest triglycerides and least favourable cholesterol ratios. These findings indicate that cardiometabolic results in athletes should be interpreted using sex, sport demands, recent workload, nutrition, and hydration rather than generic cut-offs alone. Prospective studies using standardized sampling and participant-level modelling are warranted.

Keywords: athletes; aerobic training; anaerobic training; lipid profile; urea; uric acid; sex differences

1. Introduction

Regular physical activity alters lipid transport and substrate use through changes in lipoprotein-lipase activity, triglyceride clearance, HDL remodelling, reverse cholesterol transport, mitochondrial oxidation, and body composition. Exercise interventions most consistently increase HDL-C and reduce triglycerides, while changes in total cholesterol and LDL-C are more variable and depend on exercise dose, baseline values, energy balance, and weight change (Durstine et al., 2001; Haskell et al., 2007; Kodama et al., 2007; Kraus et al., 2002; Muscella et al., 2020; Warburton et al., 2006). Consequently, the lipid profile of a trained athlete cannot be interpreted only as a static cardiovascular-risk panel; it also reflects chronic training exposure and recent metabolic conditions.

Sex adds another layer of variation. Oestrogen-related effects on hepatic lipase and lipoprotein metabolism generally contribute to higher HDL-C in premenopausal females, whereas males often present higher triglycerides and less favourable cholesterol ratios. In a large community sample, sport-related energy expenditure was associated with favourable HDL-C, LDL-C, and total cholesterol/HDL-C patterns, but the associations differed between men and women (Scheers et al., 2008). Athlete cohorts may show

additional variation because endurance, power, and intermittent sports differ in training volume, substrate demand, body composition, and recovery practices.

Urea and uric acid provide complementary information. Urea formation is influenced by protein intake, amino-acid catabolism, energy availability, hydration, renal handling, and training stress. Uric acid is the final product of purine degradation and may rise after high ATP turnover or dehydration, but it also functions as a circulating antioxidant. Neither marker is specific for overreaching or overtraining; meaningful interpretation requires serial sampling alongside training load, performance, symptoms, diet, and hydration (Hartmann & Mester, 2000; Meeusen et al., 2013; Urhausen & Kindermann, 2002). Basal biochemical values also vary by sport, age, sex, and training status (Díaz Martínez et al., 2022; Nikolaidis et al., 2003).

Previous work has largely compared active with inactive people, evaluated a single training mode, or examined males and females separately. Fewer studies have simultaneously tested sex and the predominant energy demands of sport across both lipid and nitrogen-metabolite domains in a sizeable athlete cohort. This limits the contextual interpretation of routine screening results.

Therefore, this study aimed to (1) compare lipid, urea, and uric-acid profiles among aerobic, anaerobic, and mixed-sport athletes; (2) determine sex-related differences; (3) test sex $\times$ sport-category interactions; and (4) describe the reported prevalence of values outside the study reference limits.

2. Methodology

2.1 Study design and participants

This cross-sectional secondary analysis used de-identified summary results from 429 athletes classified according to the predominant energy demands of their sports. The cohort comprised 246 males and 183 females. Aerobic sports were characterized by predominantly oxidative demands, anaerobic sports by high-intensity power and glycolytic demands, and mixed sports by substantial contributions from both systems. Exact recruitment dates, sports included, competitive level, training history, and participant age should be inserted from the original protocol.

Table 1. Participant distribution by sex and sport-energy category

Sex	Aerobic	Anaerobic	Mixed	Total
Male	130	25	91	246
Female	66	10	107	183
Total	196	35	198	429

2.2 Biochemical assessment

The cardiometabolic panel included total cholesterol, triglycerides, HDL-C, VLDL-C, LDL-C, the total cholesterol/HDL-C ratio, urea, and uric acid. Values were expressed as mg/dL except the dimensionless cholesterol ratio. Blood sample was selected from the athletes in the fasting state and collected sample at 8.00 am in the Biochemistry laboratory.

2.3 Ethics

The study protocol was reviewed and approved by the Institutional Review Board.

2.4 Statistical analysis

Data are presented as mean $\pm$ standard deviation and prevalence (%). The supplied source used one-way analysis of variance within each sex followed by least significant difference post-hoc comparisons. For the present paper, an exploratory 2×3 factorial analysis was reconstructed from the six cell sizes, means, and standard deviations for each outcome. Fixed effects were sex (male, female), sport category (aerobic, anaerobic, mixed), and their interaction. Sum-to-zero contrasts were used so that main effects represented equally weighted marginal comparisons across sport categories. F tests used 1 and 423 degrees of freedom for sex and 2 and 423 degrees of freedom for sport and interaction. Partial eta squared quantified effect magnitude. Statistical significance was set at $p < .05$. Because inputs were rounded summary statistics, all reconstructed factorial results are approximate and require confirmation using raw participant-level data. No correction for multiple outcomes was applied; therefore, interaction findings are exploratory.

3. Results

3.1 Lipid profiles

Aerobic athletes of both sexes generally displayed the most favourable lipid pattern. Male aerobic athletes had the lowest total cholesterol, triglycerides, VLDL-C, LDL-C, and total cholesterol/HDL-C ratio and the highest HDL-C. The same pattern was observed in females except that the lowest total cholesterol was also in the aerobic group and the mixed group was intermediate for most measures. Anaerobic athletes had the highest triglycerides, VLDL-C, and total cholesterol/HDL-C ratio in both sexes.

Table 2. Lipid profile of male athletes by sport-energy category

Parameter	Aerobic	Anaerobic	Mixed
Total cholesterol (mg/dL)	157.34 ± 20.39	169.04 ± 20.80	161.61 ± 19.49
Triglycerides (mg/dL)	67.35 ± 21.96	93.66 ± 26.84	67.79 ± 21.28
HDL-C (mg/dL)	49.68 ± 6.68	45.71 ± 4.46	47.37 ± 6.98
VLDL-C (mg/dL)	13.47 ± 4.39	18.73 ± 5.37	13.56 ± 4.26
LDL-C (mg/dL)	94.19 ± 21.48	104.59 ± 21.90	100.69 ± 20.04
Total cholesterol/HDL-C ratio	3.23 ± 0.65	3.70 ± 0.49	3.48 ± 0.62

Table 3. Lipid profile of female athletes by sport-energy category

Parameter	Aerobic	Anaerobic	Mixed
Total cholesterol (mg/dL)	152.17 ± 21.24	166.07 ± 15.83	163.52 ± 17.25
Triglycerides (mg/dL)	59.04 ± 25.13	77.89 ± 17.31	69.89 ± 22.67
HDL-C (mg/dL)	60.22 ± 8.91	54.38 ± 7.45	56.20 ± 8.66
VLDL-C (mg/dL)	11.81 ± 5.03	15.58 ± 3.46	13.98 ± 4.53
LDL-C (mg/dL)	80.14 ± 23.50	96.11 ± 10.71	93.35 ± 19.28
Total cholesterol/HDL-C ratio	2.61 ± 0.67	3.08 ± 0.25	2.98 ± 0.58

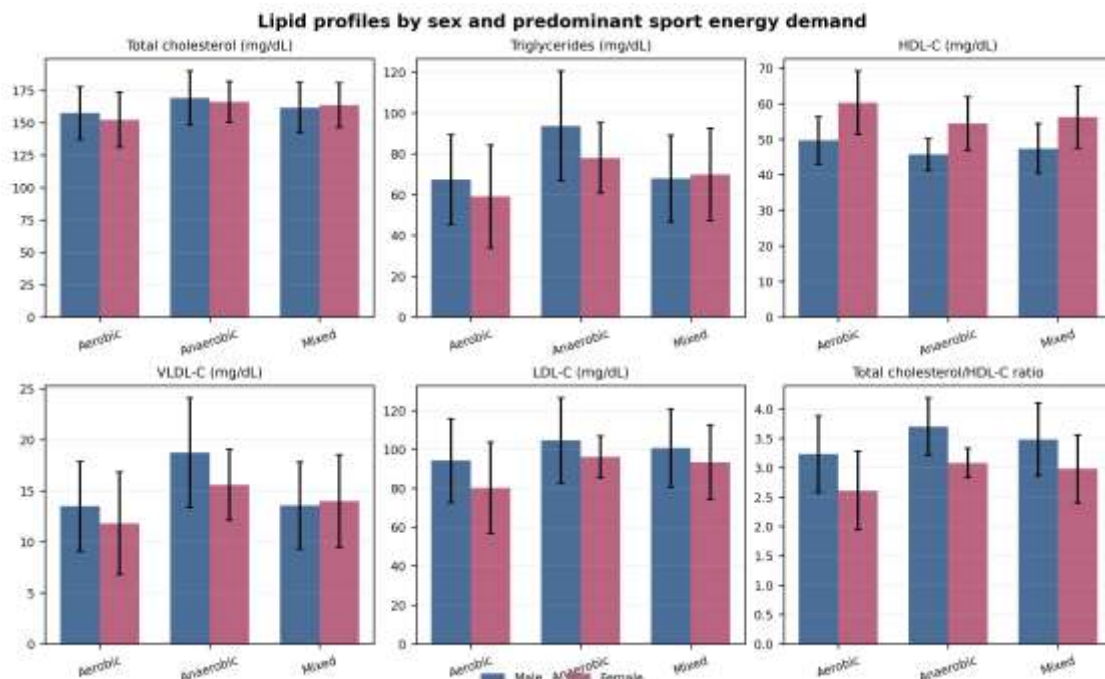


Fig 1: Lipid profiles by sex and predominant sport energy demand. Error bars represent standard deviations.

3.2 Urea and uric acid

Mean urea and uric acid were highest in aerobic athletes of both sexes. Male aerobic athletes had urea of 31.73 ± 5.73 mg/dL and uric acid of 5.37 ± 1.15 mg/dL; corresponding female values were 30.25 ± 4.75 and 4.25 ± 0.81 mg/dL. Anaerobic groups had the lowest means, with mixed-sport athletes intermediate except that female mixed-sport urea was close to the anaerobic mean.

Table 4. Metabolite profile by sex and sport-energy category

Parameter	Sex	Aerobic	Anaerobic	Mixed
Urea (mg/dL)	Male	31.73 ± 5.73	26.14 ± 5.71	29.68 ± 7.10
Urea (mg/dL)	Female	30.25 ± 4.75	27.33 ± 5.68	27.76 ± 4.71
Uric acid (mg/dL)	Male	5.37 ± 1.15	4.43 ± 1.23	5.04 ± 1.28
Uric acid (mg/dL)	Female	4.25 ± 0.81	3.54 ± 0.64	3.70 ± 0.84

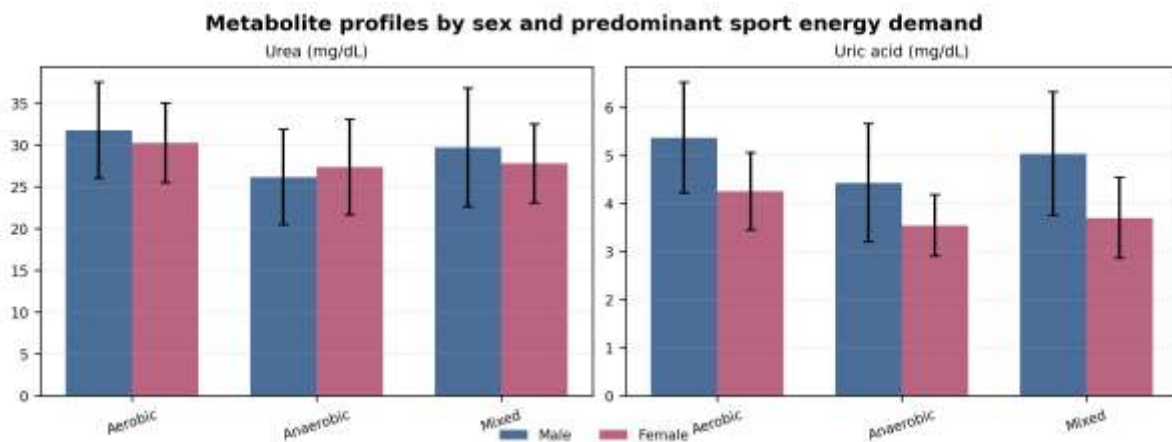


Fig 2: Urea and uric-acid profiles by sex and predominant sport energy demand. Error bars represent standard deviations.

3.3 Factorial effects of sex and sport category

Sport category showed a significant effect for every biochemical outcome (all $p < .001$), with partial eta squared values from .045 to .066. Sex effects were strongest for HDL-C (partial eta squared=.150), uric acid (.114), and the total cholesterol/HDL-C ratio (.093). Females had higher HDL-C but lower uric acid and cholesterol ratios. Significant sex $\times$ sport-category interactions were found for triglycerides and VLDL-C (both $p = .031$); the rise from aerobic to anaerobic sport was larger in males. Other interactions were not significant.

Table 5. Reconstructed two-factor analysis of cardiometabolic outcomes

Outcome	Effect	F	p	Partial eta ²
Total cholesterol	Sex	0.556	.457	0.001
	Sport category	10.022	<.001	0.045
	Sex $\times$ sport	1.543	.215	0.007
Triglycerides	Sex	5.106	.024	0.012
	Sport category	12.719	<.001	0.057
	Sex $\times$ sport	3.509	.031	0.016
HDL-C	Sex	74.939	<.001	0.150
	Sport category	10.518	<.001	0.047
	Sex $\times$ sport	0.645	.525	0.003
VLDL-C	Sex	5.093	.025	0.012

LDL-C	Sport category	12.716	<.001	0.057
	Sex × sport	3.502	.031	0.016
	Sex	11.228	<.001	0.026
TC/HDL-C ratio	Sport category	12.313	<.001	0.055
	Sex × sport	1.233	.292	0.006
	Sex	43.625	<.001	0.093
Urea	Sport category	15.028	<.001	0.066
	Sex × sport	0.475	.622	0.002
	Sex	0.825	.364	0.002
Uric acid	Sport category	11.220	<.001	0.050
	Sex × sport	0.935	.394	0.004
	Sex	54.502	<.001	0.114
	Sport category	12.116	<.001	0.054
	Sex × sport	0.853	.427	0.004

Note. Sex: $F(1, 423)$; sport and interaction: $F(2, 423)$. Statistics were reconstructed from rounded cell sizes, means, and standard deviations and must be verified using participant-level data.

3.4 Prevalence outside study reference limits

No abnormal values were reported in either anaerobic group, although the very small female anaerobic sample ($n=10$) limits interpretation. Aerobic groups generally had the highest prevalence of adverse lipid values. Female aerobic athletes showed 7.6% prevalence for high total cholesterol and low HDL-C. The highest reported single prevalence was high uric acid in male mixed-sport athletes (12.2%), but this value conflicts with 2.2% in the source narrative and requires verification.

Table 6. Reported prevalence outside study reference limits

Parameter	Sex	Aerobic	Anaerobic	Mixed
Total cholesterol	Male	6.1%	0.0%	2.8%
Total cholesterol	Female	7.6%	0.0%	0.9%
Triglycerides	Male	3.0%	0.0%	1.9%
Triglycerides	Female	4.5%	0.0%	1.9%
Low HDL-C	Male	4.6%	0.0%	3.3%
Low HDL-C	Female	7.6%	0.0%	3.7%
VLDL-C	Male	1.5%	0.0%	1.1%
VLDL-C	Female	4.5%	0.0%	2.8%
LDL-C	Male	2.3%	0.0%	2.2%
LDL-C	Female	4.5%	0.0%	0.9%
TC/HDL-C ratio	Male	3.9%	0.0%	2.2%
TC/HDL-C ratio	Female	6.1%	0.0%	3.7%
Urea	Male	5.4%	0.0%	4.0%
Urea	Female	4.5%	0.0%	3.7%
Uric acid	Male	4.6%	0.0%	12.2%
Uric acid	Female	4.5%	0.0%	1.9%



Fig 3: Reported prevalence outside study reference limits. HDL-C prevalence denotes low values; all other markers denote high values.

4. Discussion

The principal finding was that predominant sport energy demand influenced all measured lipid and nitrogen-metabolite outcomes, while sex strongly influenced HDL-C, cholesterol ratio, LDL-C, and uric acid. Aerobic athletes showed the most favourable mean lipid profile but the highest urea and uric-acid concentrations. Anaerobic athletes had higher triglycerides, VLDL-C, LDL-C, and cholesterol ratios and lower HDL-C. Importantly, effect sizes for sport category were small to moderate, demonstrating meaningful group tendencies without implying that sport category alone determines an individual athlete's biochemical status.

4.1 Lipid adaptations to sport energy demands

The higher HDL-C and lower triglycerides, VLDL-C, and cholesterol ratios observed in aerobic athletes are consistent with established endurance-training adaptations. Repeated oxidative exercise increases skeletal-muscle lipoprotein-lipase activity, triglyceride-rich particle clearance, fatty-acid oxidation, and HDL maturation and turnover (Durstine et al., 2001; Muscella et al., 2020; Sarzynski et al., 2018). Randomized evidence indicates that exercise volume is particularly important: greater weekly energy expenditure produces broader favourable lipoprotein changes even when weight loss is limited (Kraus et al., 2002; Leon et al., 2000). A meta-analysis also found a modest increase in HDL-C after aerobic training and suggested that exercise duration per session is an important determinant (Kodama et al., 2007).

The anaerobic groups' higher triglycerides and cholesterol ratios should not be interpreted as evidence that resistance or power training is unhealthy. Cross-sectional sport groups differ in body mass, muscle mass, diet, energy surplus, training phase, and recent exercise exposure. Resistance exercise can improve lipid profiles, although the magnitude and consistency of change vary across outcomes and

programmes (Kelley & Kelley, 2009; Mann et al., 2014; Tambalis et al., 2009). Moreover, mean values in all groups were generally within conventional adult clinical ranges. The present results therefore describe relative sport-group patterns rather than disease.

The sport effect for total cholesterol was significant, but its sex effect and sex $\times$ sport interaction were not. This aligns with evidence that exercise-induced changes are often clearer for HDL-C, triglycerides, particle characteristics, and ratios than for total cholesterol alone (Durstine et al., 2001; Kraus et al., 2002). Consequently, relying on total cholesterol could obscure meaningful training-related differences in lipoprotein distribution.

4.2 Sex differences and interaction effects

Females had substantially higher HDL-C and lower total cholesterol/HDL-C ratios, producing the largest sex effects in the lipid panel. This pattern is physiologically plausible given sex-hormone effects on hepatic lipase, apolipoprotein metabolism, fat distribution, and substrate utilization. Scheers et al. (2008) similarly reported sex-dependent associations between sport-related energy expenditure and HDL-C, LDL-C, and cholesterol ratios.

Triglycerides and VLDL-C showed small but significant sex $\times$ sport interactions. The anaerobic-to-aerobic difference was larger in males, suggesting that the lipid signature of sport specialization may not be identical between sexes. Because the interaction tests were reconstructed from rounded summaries, were not adjusted for multiple outcomes, and included only 10 female anaerobic athletes, they should be treated as hypothesis-generating. Raw-data models should adjust for age, body composition, dietary intake, alcohol, training volume, and menstrual or contraceptive factors.

4.3 Urea and uric acid as contextual monitoring markers

Aerobic athletes' higher urea is compatible with greater cumulative training duration, amino-acid oxidation during prolonged exercise, glycogen availability, protein intake, or lower hydration at sampling. Urea is affected by renal function and diet and cannot independently identify a catabolic or overtrained state (Baffour-Awuah et al., 2017; Banfi et al., 2012). Similarly, uric acid may rise through purine nucleotide degradation during sustained ATP turnover or through reduced plasma volume. Its interpretation is complicated by dietary purines, renal handling, sex, body composition, and antioxidant activity.

The strong sex effect for uric acid is consistent with generally lower concentrations in premenopausal females, but the study did not document reproductive or hormonal status. The sport-group differences support using urea and uric acid as contextual monitoring variables rather than diagnostic markers. Consensus guidance emphasizes that overtraining is a clinical and performance diagnosis requiring exclusion of illness and integration of workload, recovery, symptoms, and performance change; no single blood marker is sufficient (Meeusen et al., 2013; Urhausen & Kindermann, 2002). Serial individual baselines are preferable to isolated comparisons with population limits (Fallon, 2008; Halson, 2014; Hartmann & Mester, 2000).

4.4 Practical implications

Sports-science and medical teams should interpret cardiometabolic panels against sex, sport demands, body composition, dietary pattern, training phase, and sample timing. A practical monitoring approach would standardize morning fasting collection, avoid strenuous exercise for a defined pre-sampling interval, document hydration and recent diet, and compare repeated results with the athlete's own baseline. HDL-C, triglycerides, and the cholesterol/HDL-C ratio appear particularly informative for distinguishing group patterns, whereas urea and uric acid may help interpret energy availability, protein turnover, and hydration only when combined with contextual data.

4.5 Strengths and limitations

Strengths include the relatively large cohort, inclusion of both sexes, simultaneous analysis of lipid and metabolite domains, and classification by sport energy demand. The factorial framework separates sex and sport effects and highlights potential interactions that are hidden in sex-stratified one-way comparisons.

The cross-sectional design prevents causal inference. Group sizes were unequal, particularly the female anaerobic group ($n=10$). Broad sport categories are proxies for habitual metabolic demand and do not measure individual training. Age, body composition, diet, hydration, alcohol, supplements, renal function, menstrual characteristics, training phase, recent exercise, and laboratory procedures were unavailable. Factorial statistics were reconstructed from rounded summary data; assumptions, outliers, covariate adjustment, and exact confidence intervals could not be assessed. Multiple outcomes were tested

without multiplicity correction. Finally, the abnormal-value thresholds and the male mixed-sport uric-acid prevalence require confirmation from the raw dataset.

5. Conclusion

Sex and predominant sport energy demand were associated with distinct cardiometabolic profiles. Aerobic athletes exhibited higher HDL-C and lower triglycerides, VLDL-C, LDL-C, and cholesterol ratios, but higher urea and uric acid. Females showed higher HDL-C and lower cholesterol ratios and uric acid than males, while triglyceride and VLDL-C sport patterns differed modestly by sex. These findings support sex- and sport-contextualized interpretation of athlete blood tests and serial monitoring under standardized conditions. Confirmation using participant-level data and prospective tracking of training, nutrition, hydration, body composition, and performance is required.

References

- Banfi, G., Colombini, A., Lombardi, G., & Lubkowska, A. (2012). Metabolic markers in sports medicine. *Advances in Clinical Chemistry*, 56, 1-54. <https://doi.org/10.1016/B978-0-12-394317-0.00015-7>
- Baffour-Awuah, B., Addai-Mensah, O., Moses, M. O., Mensah, W., Ibekwe, B. C., Essaw, E., & Acheampong, I. K. (2017). Differences in haematological and biochemical parameters of athletes and non-athletes. *Journal of Advances in Medicine and Medical Research*, 24(12), 1-5. <https://doi.org/10.9734/JAMMR/2017/38217>
- Díaz Martínez, A. E., Alacid, F., Martínez, I., & Muyor, J. M. (2022). Basal values of biochemical and hematological parameters in elite athletes. *International Journal of Environmental Research and Public Health*, 19(5), 3059. <https://doi.org/10.3390/ijerph19053059>
- Durstine, J. L., Grandjean, P. W., Davis, P. G., Ferguson, M. A., Alderson, N. L., & DuBose, K. D. (2001). Blood lipid and lipoprotein adaptations to exercise: A quantitative analysis. *Sports Medicine*, 31(15), 1033-1062. <https://doi.org/10.2165/00007256-200131150-00002>
- Fallon, K. E. (2008). The clinical utility of screening of biochemical parameters in elite athletes: Analysis of 100 cases. *British Journal of Sports Medicine*, 42(5), 334-337. <https://doi.org/10.1136/bjsm.2007.041137>
- Halson, S. L. (2014). Monitoring training load to understand fatigue in athletes. *Sports Medicine*, 44(Suppl 2), S139-S147. <https://doi.org/10.1007/s40279-014-0253-z>
- Hartmann, U., & Mester, J. (2000). Training and overtraining markers in selected sport events. *Medicine & Science in Sports & Exercise*, 32(1), 209-215. <https://doi.org/10.1097/00005768-200001000-00031>
- Haskell, W. L., Lee, I.-M., Pate, R. R., Powell, K. E., Blair, S. N., Franklin, B. A., Macera, C. A., Heath, G. W., Thompson, P. D., & Bauman, A. (2007). Physical activity and public health: Updated recommendation for adults. *Medicine & Science in Sports & Exercise*, 39(8), 1423-1434. <https://doi.org/10.1249/mss.0b013e3180616b27>
- Kelley, G. A., & Kelley, K. S. (2009). Impact of progressive resistance training on lipids and lipoproteins in adults: A meta-analysis of randomized controlled trials. *Preventive Medicine*, 48(1), 9-19. <https://doi.org/10.1016/j.ypmed.2008.10.010>
- Kodama, S., Tanaka, S., Saito, K., Shu, M., Sone, Y., Onitake, F., Suzuki, E., Shimano, H., Yamamoto, S., Kondo, K., Ohashi, Y., Yamada, N., & Sone, H. (2007). Effect of aerobic exercise training on serum levels of high-density lipoprotein cholesterol: A meta-analysis. *Archives of Internal Medicine*, 167(10), 999-1008. <https://doi.org/10.1001/archinte.167.10.999>
- Kraus, W. E., Houmard, J. A., Duscha, B. D., Knetzger, K. J., Wharton, M. B., McCartney, J. S., Bales, C. W., Henes, S., Samsa, G. P., Otvos, J. D., Kulkarni, K. R., & Slentz, C. A. (2002). Effects of the amount and intensity of exercise on plasma lipoproteins. *New England Journal of Medicine*, 347(19), 1483-1492. <https://doi.org/10.1056/NEJMoa020194>
- Leon, A. S., Rice, T., Mandel, S., Després, J.-P., Bergeron, J., Gagnon, J., Rao, D. C., Skinner, J. S., & Bouchard, C. (2000). Blood lipid response to 20 weeks of supervised exercise in a large biracial population. *Metabolism*, 49(4), 513-520. [https://doi.org/10.1016/S0026-0495\(00\)80018-4](https://doi.org/10.1016/S0026-0495(00)80018-4)

- Mann, S., Beedie, C., & Jimenez, A. (2014). Differential effects of aerobic exercise, resistance training and combined exercise modalities on cholesterol and the lipid profile: Review, synthesis and recommendations. *Sports Medicine*, 44(2), 211-221. <https://doi.org/10.1007/s40279-013-0110-5>
- Meeusen, R., Duclos, M., Foster, C., Fry, A., Gleeson, M., Nieman, D., Raglin, J., Rietjens, G., Steinacker, J., & Urhausen, A. (2013). Prevention, diagnosis, and treatment of the overtraining syndrome: Joint consensus statement. *Medicine & Science in Sports & Exercise*, 45(1), 186-205. <https://doi.org/10.1249/MSS.0b013e318279a10a>
- Muscella, A., Stefàno, E., & Marsigliante, S. (2020). The effects of exercise training on lipid metabolism and coronary heart disease. *American Journal of Physiology-Heart and Circulatory Physiology*, 319(1), H76-H88. <https://doi.org/10.1152/ajpheart.00708.2019>
- Nikolaidis, M. G., Protosygiellou, M. D., Petridou, A., Tsalis, G., Tsigilis, N., & Mougios, V. (2003). Hematologic and biochemical profile of juvenile and adult athletes of both sexes: Implications for clinical evaluation. *International Journal of Sports Medicine*, 24(7), 506-511. <https://doi.org/10.1055/s-2003-42014>
- Sarzynski, M. A., Ruiz-Ramie, J. J., Barber, J. L., Slentz, C. A., Apolzan, J. W., McGarrah, R. W., Harris, M. N., Church, T. S., Borja, M. S., He, Y., Wang, T., & Kraus, W. E. (2018). Effects of increasing exercise intensity and dose on multiple measures of HDL function. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 38(4), 943-952. <https://doi.org/10.1161/ATVBAHA.117.310307>
- Scheers, T., Philippaerts, R., Lefevre, J., Van Langendonck, L., Duquet, W., Thomis, M., & Beunen, G. (2008). Lipid profile in men and women with different levels of sports participation and physical activity. *Public Health Nutrition*, 11(11), 1098-1106. <https://doi.org/10.1017/S1368980007001425>
- Tambalis, K., Panagiotakos, D. B., Kavouras, S. A., & Sidossis, L. S. (2009). Responses of blood lipids to aerobic, resistance, and combined aerobic with resistance exercise training: A systematic review of current evidence. *Angiology*, 60(5), 614-632. <https://doi.org/10.1177/0003319708324927>
- Urhausen, A., & Kindermann, W. (2002). Diagnosis of overtraining: What tools do we have? *Sports Medicine*, 32(2), 95-102. <https://doi.org/10.2165/00007256-200232020-00002>
- Warburton, D. E. R., Nicol, C. W., & Bredin, S. S. D. (2006). Health benefits of physical activity: The evidence. *Canadian Medical Association Journal*, 174(6), 801-809. <https://doi.org/10.1503/cmaj.051351>