

Commentary

Occupational Factors, Fatigue, and Cardiovascular Disease

Sean Collins, PT, ScD, CCS

Chairperson & Associate Professor, Department of Physical Therapy, Research Associate,
Kerr Ergonomics Institute, University of Massachusetts Lowell

ABSTRACT

Purpose: Briefly identify the epidemiological evidence, propose pertinent mechanisms, and discuss physical therapy practice as well as research implications of a causal association between occupational factors and cardiovascular disease. **Summary of Key Points:** There is evidence that occupational metabolic demands and work organizations characterized by reduced worker control are associated with increased risk of cardiovascular disease. It is biologically plausible that these two factors interact to create a preclinical, intermediate state of fatigue (burnout) that is a critical component in the causal path from occupational factors to CVD. Physical therapists are uniquely qualified to contribute to an understanding of these mechanisms and their resultant implications for work organization, rehabilitation, and health promotion. **Statement of Recommendations:** Physical therapists engaged in ergonomic job analysis should consider work related metabolic demands, worker control, and fatigue in their assessment of risk for injury and illness, in recommendations for return to work, and in the prescription of health promotion leisure time physical activity

Key Words: work, heart disease

INTRODUCTION AND PURPOSE

This paper will identify the epidemiological evidence for a causal association between 2 specific occupational factors and cardiovascular disease (CVD)—high metabolic demands and work organizations characterized by low worker control. These 2 factors have been chosen based on a proposed interactive relationship with CVD through the common preclinical physiological pathway of fatigue. Fatigue will be considered a cluster of signs and symptoms associated with chronically reduced physiological capacity for responding to the continued energetic and regulatory demands of goal-directed activity. While not the purpose of this paper, in addition to increasing risk for CVD, fatigue in occupational, home, and leisure activities can reduce performance in a variety of ways, as well as increasing subjective health complaints and risk of injuries.¹⁻³

The paper is organized into 3 primary sections. The first section, occupational factors, will define and identify

available evidence linking these factors to fatigue and to CVD. The second section will propose a mechanistic pathway through which increased occupational metabolic demands when combined with reduced worker control results in fatigue; and fatigue, through its physiological components, increases risk for CVD. The third section will introduce physical therapy practice and research implications of the proposed mechanistic pathway.

OCCUPATIONAL FACTORS THAT MAY AFFECT DEVELOPMENT OF CVD

Physical Demands of Work

The development of mechanized and automated work processes has reduced the prevalence and severity of highly metabolically demanding work, however such demands (either consistent or intermittent) remain a required component to some types of work.⁴ There is little doubt regarding the beneficial effects of leisure time physical activity on cardiovascular risk – through a variety of pathways (vascular health, hormone regulation, immunological function, etc.). However, much of the evidence establishing this relationship has not included consideration for potentially differential effects of occupational metabolic demands and leisure time physical activity.⁵ Consideration for occupational physical activity requires quantification of the work-related metabolic demand. Such studies collect data separately on leisure time activity and work tasks. For example, Krause et al⁶ assessed energy expenditure at work using predicted metabolic equivalents for work tasks, and leisure time physical activity by self-report of hours spent exercising. When occupational physical activity has been considered separately the results have been mixed with some studies demonstrating reduced CVD risk,⁷ some demonstrating no effect,⁸ and some demonstrating an increase in risk.⁹ Studies demonstrating protective or no effect tended to measure metabolic demands with no account for worker aerobic capacity, did not control for other occupational factors (such as work organization), did not adjust for changing occupational metabolic demands over time, and did not consider preclinical signs of atherosclerosis progression (ie, carotid artery intimal thickening which is less likely to suffer from healthy worker effect^{1a}) or intermediary steps such as fatigue.

Metabolic demand exceeding approximately 35% of aerobic capacity has been proposed as a source of work related fatigue,⁴ and laboratory studies confirm that prolonged work at

^{1a}Healthy worker effect: In occupational cohorts, waiting for a chronic disease such as CVD to develop tends to lead to workers that develop the disease to be lost to follow up leaving only healthy workers remaining which biases results toward the null hypothesis.

Address correspondence to: Sean Collins, University of Massachusetts Lowell, 3 Solomont Way, Suite 5, Lowell, MA 01854 (sean_collins@uml.edu).

this low relative intensity is associated with physiological signs and subjective reports of fatigue when performed for lengths of time commensurate with a workday.¹⁰ Garbage collection is an example of an occupation where metabolic demands frequently exceed 35% of aerobic capacity,¹¹ and such work has also been demonstrated to lead to physiological changes (parasympathetic nervous system withdrawal) and subjective complaints of fatigue over time.¹²

The prevalence of metabolic demands exceeding 35% of aerobic capacity has recently been estimated at 27% of men and 22% of women in a typical working day.¹³ While these estimates are based on a small sample (94 men, 94 women), the sample was randomly drawn from a much larger occupational health cohort (8000 employees across 80 occupations), which increases its likelihood of providing accurate population estimates. A recent population-based longitudinal study, demonstrated an association between demands and progression of atherosclerosis (as assessed by carotid intimal thickening).⁶ This study controlled for several known CVD risk factors, including leisure time physical activity and other occupational factors such as stress from deadlines, and mental strain.

Work Organization

The same mechanization and automation that has reduced absolute metabolic demands of work has also changed work organization by reducing worker control of the work process. Substantial research now exists relating work organization to cardiovascular disease, with several recent reviews and large prospective studies concluding that there is a relationship between features of work organization and CVD and its major risk factors such as hypertension.¹⁴ The most consistent work organization feature associated with CVD is low job control.^{14,15} Low control was introduced as a feature of work organization related behavior and health consequences in 1979 by Karasek.¹⁶ An important conceptual feature of low job control is that it is external to the worker as opposed to an individual characteristic.¹⁷ This perspective emerges from the occupational health tradition whereby exposure sources are distinct from measuring biological and/or behavioral response to exposure.¹⁸ In occupations with low control, a worker has limited decision making authority over how, when, or where their work tasks are performed—which includes the skills used, pace, intensity, and loads. In addition to an association between low control and CVD, there has been consistent evidence that low worker control (even when adjusting for other factors) is associated with increased rates of fatigue as well as physiological markers of fatigue.¹⁹

A PROPOSED MECHANISTIC PATHWAY

The mechanistic pathway being proposed in this paper is rather simple, but yet has not been well studied. This proposal suggests that an intermediate step in the pathway from increased occupational metabolic demands to CVD includes the development of fatigue and is based on 3 assumptions: (1) occupational metabolic demands exceeding ~35% of worker aerobic capacity [metabolic demand/aerobic capacity index (MD/AC index)] leads to fatigue; (2) low worker

control increases the likelihood that high MD/AC index will result in fatigue; and (3) fatigue increases risk for CVD.

The support for assumption 1 is biologically plausible and is supported by empirical evidence as well as expert opinion.^{4,10} The primary support for assumption 2 is from the literature on the demand control model (DCM) of occupational stress. The DCM associates overall demand (psychological as well as physical) and low control with fatigue and CVD risk.^{15,19} Assumption 2 is appealing from a common sense perspective as well. If a worker under a situation of high MD/AC index has the control to modify pace, intensity, load, and rest periods, then such a worker has control to modify exposure and therefore reduce metabolic demands of an activity to restore balance and recover from periods of exertion.²⁰ Control in this regard may result in the fundamental physiological difference between metabolic demands leading to fatigue, and metabolic demands associated with leisure time physical activity and exercise resulting in adaptation. There are 2 possible trajectories of adaptation associated with exposure to physical demands. One trajectory is hypertrophy adaptation which increases resilience and capacity to withstand demands; and the second is injury which in the situation of metabolic demands could be fatigue.²¹ The modifying role of job control over which trajectory physical exposures take has been identified for musculoskeletal repetitive strain symptoms.²² When physical exposure to poor keyboard height is combined with low control there is an increase in upper extremity symptoms of repetitive strain. If these same physical exposures exist with high control, there is no increase in upper extremity symptoms.

Support for assumption 3 exists in a variety of forms due to the variety of disciplines and approaches of conceptualizing fatigue. Fatigue, a cluster of signs and symptoms associated with a chronically reduced physiological capacity for responding to the continued energetic and regulatory demands of goal-directed activity, has been conceptualized as burnout,²³ vital exhaustion,²⁴ and overtraining.²⁵ There are several reviews that highlight the many physiological consequences of fatigue herein conceptualized; those that are most clearly associated with CVD risk,²⁶ and that show physiological changes that are associated with increased CVD such as elevated cortisol levels and reduced heart rate variability.²⁷

PHYSICAL THERAPY IMPLICATIONS

There are several implications for physical therapy practice and research. Most directly, physical therapists engaged in ergonomic job analysis and employment recommendations for return to work should consider work related metabolic demands, worker aerobic capacity and worker control in their assessment of risk for injury and illness. Due to the interactive associations of these components, it is difficult to predict risk without an understanding of each component. It is equally difficult to identify proper solutions. Often, a mixed solution that involves changing tasks to reduce their demand, exercise to increase worker aerobic capacity, and participatory processes to increase worker control will have produce the most favorable reduction in risk for promotion of health.¹⁸

Physical therapists engaged in the prescription of leisure time physical activity for working populations should also consider occupational MD/AC index, job control, and fatigue in their exercise prescription. If a worker is already fatigued from chronic overexertion – the initial prescription should be rest, or perhaps relaxation-oriented exercise (yoga, light stretching, etc). Avenues to change the work demands, even if temporarily, to allow recovery should be sought. If permanent changes to either occupational metabolic demands or worker control are impossible, then once a worker recovers s/he should undergo physical conditioning with appropriate rest periods. This should allow an adaptive training response aimed at increasing aerobic capacity so that MD/AC index is < 35%.

Finally, the research methods associated with the concepts described in this paper could help identify an appropriate balance between daily living metabolic demands and aerobic capacity for sustained stability^{2a} in the home and/or community environment. It is possible that patients with reduced endurance are required, by the metabolic demands of participation in activities of daily living, to exceed a recommended MD/AC index of for the time they must participate in such activities. If this is true, fatigue could require cessation of necessary ADLs, or result in a decrement in their performance. Either scenario can lead to recurrent hospital admission and variation of safety and independence that cycles with time. Quantification of aerobic capacity is common in physical therapy practice. However, quantification of the daily demands and the MD/AC index, or utilization of newer methodologies for the examination of fatigue (cortisol, heart rate variability) may not be as common a practice.

CONCLUSION

Relationships exist between increased occupational metabolic demands, reduced job control, fatigue, and CVD. A proposed mechanistic pathway states that demands which exceed capacity, when combined with reduced control lead to fatigue which, in turn, increases risk for CVD. Physical therapists are uniquely qualified to evaluate and intervene with the interacting concepts of the proposed pathway: decreasing environmental demands, improving individual capacity, and fostering control over action as a potent means to decrease fatigue. In light of the pathway suggested, there are immediate implications for practice as well as possibilities for new directions to research.

ACKNOWLEDGEMENTS

Support for this work was provided, in part, by the Kerr Ergonomics Institute at the University of Massachusetts Lowell, through support of the author during his dissertation; and by funding from WorkWell Systems Inc, for the project: "Prediction & Measurement of Metabolic Demand & Capacity for Occupational Tasks."

^aSustained stability refers to the ability to sustain the necessary activities for being in homeostasis. Being able to sustain the ability to get up and get a drink is a critical component to maintaining hydration. If fatigue from previous mobilizations prevents someone from mobilizing to get another drink and dehydration ensues, then they are not sustaining stability.

REFERENCES

1. Hancock P, Szalma J, eds. Performance Under Stress: Ashgate; 2008. Harris D, ed. *Human Factors in Defence*.
2. Sluiter JK, de Croon EM, Meijman TF, Frings-Dresen MHW. Need for recovery from work related fatigue and its role in the development and prediction of subjective health complaints. *Occup Environ Med*. 2003;60 Suppl 1:i62-70.
3. Swaen GMH, van Amelsvoort L, Bultmann U, Kant IJ. Fatigue as a risk factor for being injured in an occupational accident: results from the Maastricht Cohort Study. *Occup Environ Med*. 2003;60(90001).
4. Åstrand PO. Textbook of Work Physiology: *Physiological Bases of Exercise*. Human Kinetics; 2003.
5. Berlin JA, Colditz GA. A meta-analysis of physical activity in the prevention of coronary heart disease. *Am J Epidemiol*. 1990;132(4):612-628.
6. Krause N, Brand RJ, Kaplan GA, et al. Occupational physical activity, energy expenditure and 11-year progression of carotid atherosclerosis. *Scand J Work Environ Health*. 2007;33(6):405.
7. Hu G, Jousilahti P, Borodulin K, et al. Occupational, commuting and leisure-time physical activity in relation to coronary heart disease among middle-aged Finnish men and women. *Atherosclerosis*. 2007;194(2):490-497.
8. Haapanen N, Miilunpalo S, Vuori I, Oja P, Pasanen M. Characteristics of leisure time physical activity associated with decreased risk of premature all-cause and cardiovascular disease mortality in middle-aged men. *Am J Epidemiol*. 1996;143(9):870-880.
9. Kristal-Boneh E, Harari G, Melamed S, Froom P. Association of Physical Activity at Work With Mortality in Israeli Industrial Employees: The CORDIS Study. *J Occup Environ Med*. 2000;42(2):127.
10. Wang M-JJ. Relationship between maximum acceptable work time and physical workload. *Ergonomics*. 2002;45(4):280-289.
11. Anjos LA, Ferreira JA, Damião JJ. Heart rate and energy expenditure during garbage collection in Rio de Janeiro, Brazil. *Cadernos de Saúde Pública*. 2007;23:2749-2755.
12. Pichot V, Bourin E, Roche F, et al. Quantification of cumulated physical fatigue at the workplace. *Pflügers Arch Eur J Physiol*. 2002;445(2):267-272.
13. Karlqvist L, Leijon O, Härenstam A. Physical demands in working life and individual physical capacity. *Eur J Appl Physiol*. 2003;89(6):536-547.
14. Belkic KL, Landsbergis PA, Schnall PL, Baker D. Is job strain a major source of cardiovascular disease risk? *Scand J Work Environ Health*. 2004;30(2):85-128.
15. Karasek R. Low social control and physiological deregulation—the stress–disequilibrium theory, towards a new demand–control model. *SJWEH*. 2008.
16. Karasek R. Job demands, job decision latitude and mental strain: Implications for job redesign. *Adm Sci Q*. 1979;24:285-308.
17. Karasek R, Theorell T. *Healthy Work: Stress Productivity, and the Reconstruction of Working Life*. New York, NY: Basic Books; 1990.

18. Collins S, Landsbergis P, Warren N, LaMontagne AD. Stopping stress at its origins: addressing working conditions. *Hypertension*. 2007;49(5):e33.
19. Collins SM, Karasek RA, Costas K. Job strain and autonomic indices of cardiovascular disease risk. *Am J Ind Med*. 2005;48(3):182-193.
20. Sonnentag S, Zijlstra FRH. Job characteristics and off-job activities as predictors of need for recovery, well-being, and fatigue. *J Appl Psychol*. 2006;91(2):330-349.
21. Mueller MJ, Maluf KS. Tissue adaptation to physical stress: a proposed "Physical Stress Theory" to guide physical therapist practice, education, and research. *Phys Ther*. 2002;82(4):383-403.
22. Faucett J, Rempel D. VDT-related musculoskeletal symptoms: interactions between work posture and psychosocial work factors. *Am J Ind Med*. 1994;26(5).
23. Maslach C, Jackson SE. The measurement of experienced burnout. *J Occup Behav*. 1981:99-113.
24. Appels A. Exhausted subjects, exhausted systems. *Physiologica Scandinavica*. 1997;161:153-154.
25. Kentta G, Hassmen P. Overtraining and recovery. A conceptual model. *Sports Med*. 1998;26(1):1-16.
26. Melamed S, Shirom A, Toker S, Berliner S, Shapira I. Burnout and risk of cardiovascular disease: evidence, possible causal paths, and promising research directions. *Psychol Bull*. 2006;132(3):327-353.
27. Mourot L, Bouhaddi M, Perrey S, et al. Decrease in heart rate variability with overtraining: assessment by the Poincare plot analysis. *Clin Physiol Funct Imaging*. 2004;24(1):10-18.

**Franklin Pierce University
College of Graduate and Professional Studies
Doctor of Physical Therapy Program**



Franklin Pierce University is seeking to expand our faculty in our CAPTE approved expansion program in Goodyear, Arizona. Join our new dynamic faculty team who are responsible for teaching the first year curriculum and become a faculty member teaching the second year curriculum.

**Full-time Faculty needed starting May 2010:
Arizona Licensed Physical Therapist with the following qualifications:**

- Cardiovascular and pulmonary specialization (CCS) and/or Neurologic specialization (NCS)
- Terminal degree (preferred) or Advanced Masters (considered) with strong cardiac or pulmonary rehabilitation and/or neurologic background, including clinical expertise in treatment of adults in the Acute Care, Acute Rehabilitation or Skilled Nursing Facility environments.
- Teaching experience in an entry-level physical therapy program.

Teaching assignment: PTH504 Pathophysiology of Cardiopulmonary System & Muscles, Professional Year 1, Term 3
PTH513 Physical Therapy Practice II-Adults, Professional Year 2, Term 1 – and/or – PTH509 Neuroscience II, Professional Year 2, Term 1
Applicants should send a letter of intent and curriculum vitae to:

Human Resources
Franklin Pierce University
40 University Drive
Rindge, NH 03461

Or – fax to 603-899-4326, online @ www.franklinpierce.edu or via email to www.hrdept@franklinpierce.edu