学校法人 香川栄養学園 | 80th

香川栄養学園創立 80 周年記念事業プレイベント

最新の栄養学と調理実習で
家庭科教諭のスキルアップ！

卒業生家庭科教諭のための 80 周年記念事業運営委員会
顧問　　香川芳子（学長）
委員長　五明紀春（副学長）

平成 24 年 8 月 5 日(日)　9:30ー16:00
女子栄養大学坂戸キャンパス

本学出身家庭科教員の皆様　お帰りなさい。

本学では短大創設以来、学園の建学の精神からも大切な資格として
家庭科教員の養成に努め、また、文部科学省からも中央研修をお引き受け
してきました。

特に献立作成の食品構成は本学のものが使用されてきたことは誇りです。

今回80周年記念事業プレイベントに大勢お集まりになるとのこと、お目に
かかるのを楽しみにしています。

ご報告など沢山お聞かせ下さい。

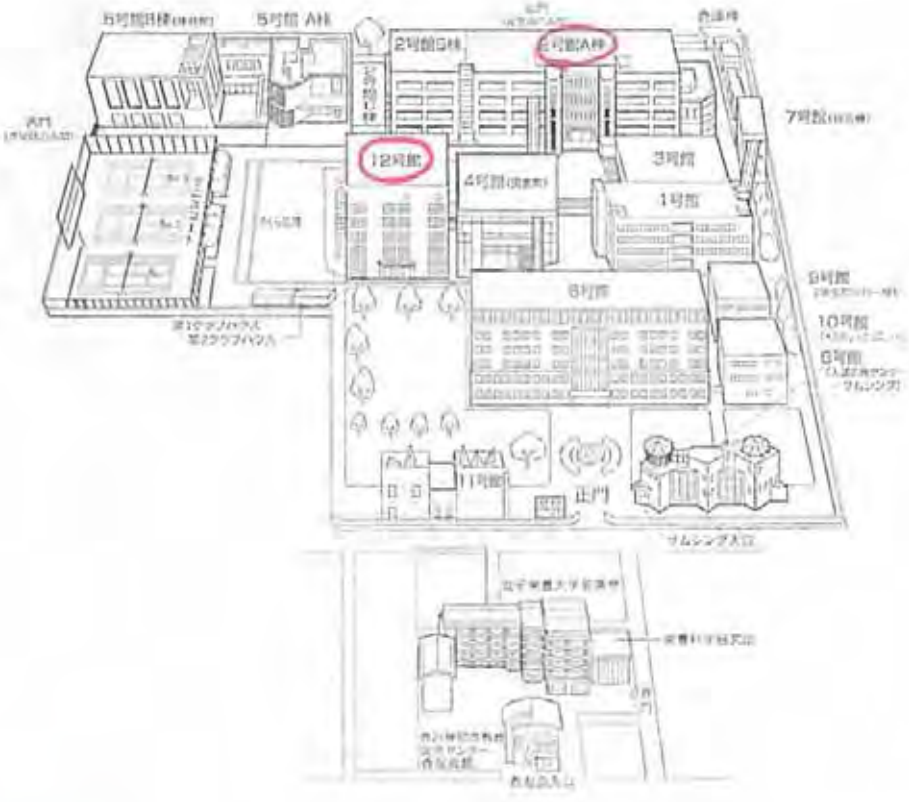
女子栄養大学
女子栄養大学短期大学部
学長　香川芳子

プログラム

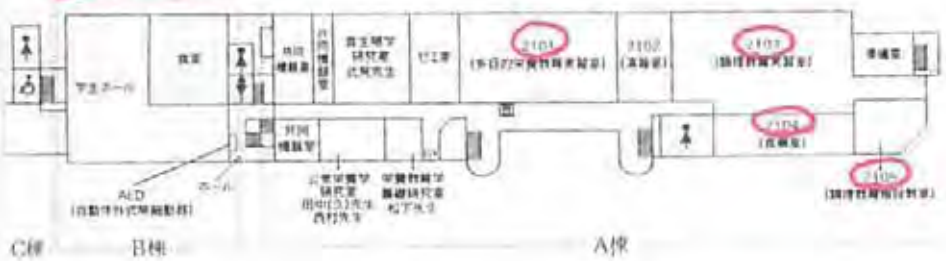
時間	プログラム	会場
9:30～ 9:40	開会のことば　　学長　香川芳子先生	12 号館 3 階　12301 講義室
9:40～10:00	総会（今後のネットワークづくり等） 運営委員会委員長　五明紀春先生 プロジェクトリーダー　井元りえ	同上
10:00～11:00	特別講演「時間栄養学」 副学長　香川靖雄先生	同上
11:15～14:15	調理実習 准教授　松田康子先生 助教　　駒場千佳子先生	2 号館 1 階 2105 調理教育階段教室 2103 調理教育実習室 2104 食事室
14:30～15:00	ベテラン家庭科教諭による 授業実践のお話 杉 信子 先生 1975（昭和 50）年（学部）卒 ご勤務先：横浜英和女学院中学高等学校 原 奈都子先生 1979（昭 54）年（短大）卒 ご勤務先：江戸川区立小松川第二中学校）	2105 調理教育階段教室
15:00～16:00	情報交換会「家庭科 Education Cafe」	2101 多目的栄養教育実習室

坂戸キャンパスマップ

12号館3階



2号館1階



「女子栄養大学家庭科ネットワークの会」規約

第1条（名称）本会は、「女子栄養大学家庭科ネットワークの会」と称する。

第2条（目的）本会は、女子栄養大学・女子栄養短期大学で家庭科教員免許を取得した人と、女子栄養大学の教職員、及び家庭科教諭を目指す在学生が、情報交換を行うことにより、交流を深め、相互啓発することを目的とする。

第3条（事業）本会は、大学のネット環境を用いた情報交換活動（授業実践の内容や方法、教員採用情報、高校生の進学希望などを含む）、及び研究会・セミナーの開催などの事業を行う。

第4条（会員）本会員は、次の通りとする。

(1) 免許取得会員：女子栄養大学・女子栄養短期大学で家庭科教員免許を取得した人。
(2) 学生会員：女子栄養大学で家庭科教員免許の取得を目指す学生。
(3) 教職員会員：本会の目的に賛同し、卒業生や学生への助言・協力を行う現職および元教職員。
(4) 本会の目的に賛同する人。

第5条（会長）本会の会長は、女子栄養大学学長とする。

第6条（各種活動組織）本会は、本会の目的に沿った各種の活動を行う会を組織する。

第7条（事務局）本会の事務局は、教職課程・家庭科委員会とする。

本会則は、平成24年8月5日より施行する。

大学の e-learning システム、'Course Power' を用いた情報交換について

1. 機 能

「女子栄養大学家庭科ネットワークの会」のクラスができました。

それを利用して、様々な情報交換を行いたいと思います。

(1) 大学が提供する資料を参照したり、ダウンロードすることができます。

(2) 掲示板（フォーラム）機能を用いて、文章、添付ファイル、画像などを投稿することにより、授業実践の情報などを相互交換できます。

2. 方 法

・eiyo アドレスとパスワードで、このクラスに入ることができます。

・ 本学の情報・ネットワーク担当が、eiyo アドレスとパスワードをつくりますので、別紙に記入し、ご提出ください。

・ eiyo アドレスとパスワードの準備ができましたら、使い方説明と共に郵送いたします。

・ご活用の程、どうぞよろしくお願い致します。

特別講演　「時間栄養学」

副学長　香川靖雄 先生

女子栄養大学学園創立80周年記念家庭科教諭
記念講演「時間栄養学」 於12301教室
2012年8月5日(日)10:10-11:10 女子栄養大学 副学長 香川靖雄

朝食を充実。野菜先摂取。朝:昼:夕=3:3:4のｴﾈﾙｷﾞｰ配分に。21時以降は軽食

家庭科教育の新たな指針として、時間栄養学が重視されるようになった。時計遺伝子とﾃﾛﾒｱの発見は、栄養素の種類、摂取量による従来の家庭科教諭の常識を大きく変革した。時間栄養学によって、心身活動の基盤となる家庭生活が改善される。どのような朝、昼、夕の割合で食物をどの時刻、順、速度で摂取するかは精神と代謝に大きく影響する。時間栄養学により精神労働を中心とする現代社会の学校の知育、体育、徳育の基盤が形成される。

香川靖雄編:日本栄養・食糧学会監修「時間栄養学」女子栄養大学出版部

1

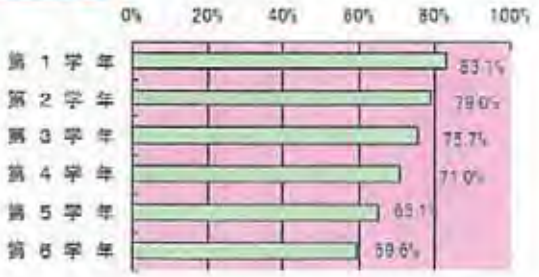
健康日本21の栄養の15目標で朝食欠食とｶﾙｼｳﾑ摂取は特に悪化D○。適正体重や脂肪ｴﾈﾙｷﾞｰ比は不変C、やや改善したのは理解。目標値に達したのはﾒﾀﾎﾞを知っている人の割合だけA○。

2

早寝早起きは高学年で6割に低下

埼玉県調査

早寝、早起きをしていますか　□している

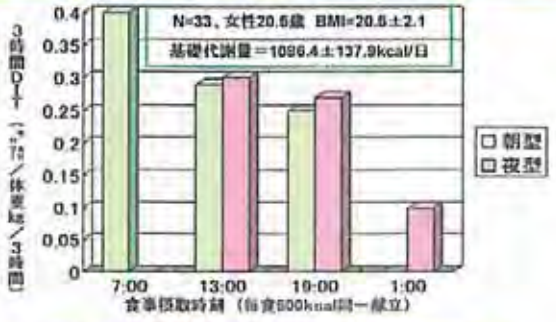


3

時間栄養学は栄養科学部門でﾍﾞｽﾄｾﾗｰ1位

4

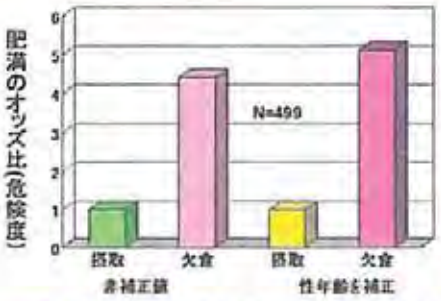
同一カロリー同一献立でも食事誘発性熱産生は4倍も異なる。朝は心身の活動に、夜は肥満に消費



間野由香他 日本栄養・食糧学会誌63:101-106 (2010)

5

朝食欠食による肥満の頻度予測値



Ma Y. et al. Am J.Epidemiol 158:85-92(2003)

6

遠赤外線サーモグラフィーによる体表温度画像表示

朝食欠食で人体はエネルギー節約反応

朝食摂取前の低代謝活性

図2. 朝食による全身代謝の活性化

30

朝食摂取で内臓時計遺伝子活発化

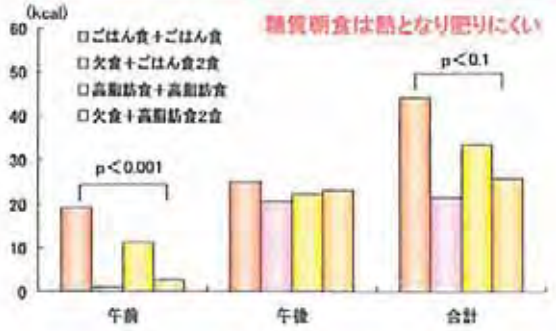
朝食摂取後の心身代謝の活性化

34

香川靖雄：第48回アジア太平洋栄養士会議生涯教育講演会抄録集より 2007年11月24日

7

食事パターンによる食事誘発性熱産生の比較

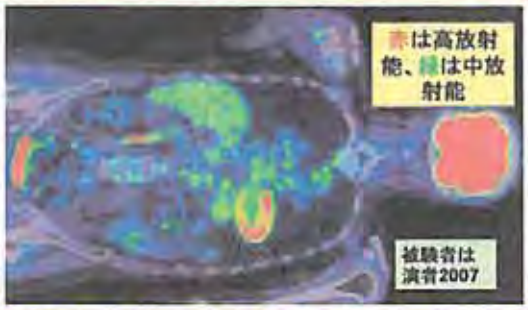


糖質朝食は脂となり肥りにくい

永井成美、坂根直樹、森谷敏雄：糖尿病 48：761-770 (2005)

8

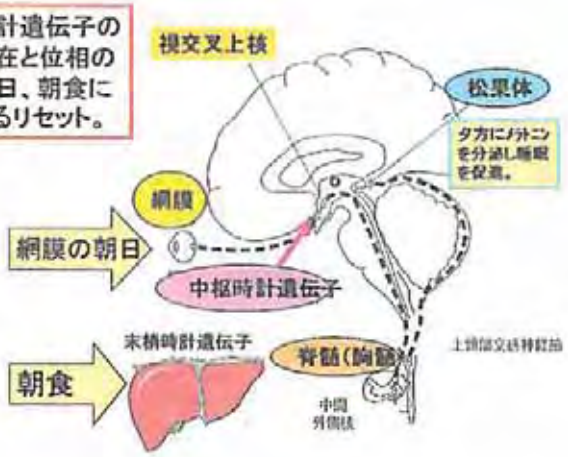
朝食欠食後は脳にはグルコース代謝が集中して脳の活動の維持を図る。血糖低下で脳機能低下。



^{18}F-デオキシグルコース投与後の陽子CT像　脳、心臓、肝臓の活発な糖代謝

9

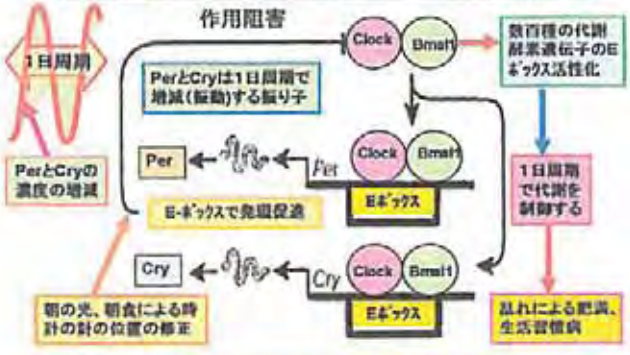
時計遺伝子の局在と位相の朝日、朝食によるリセット。



10

11

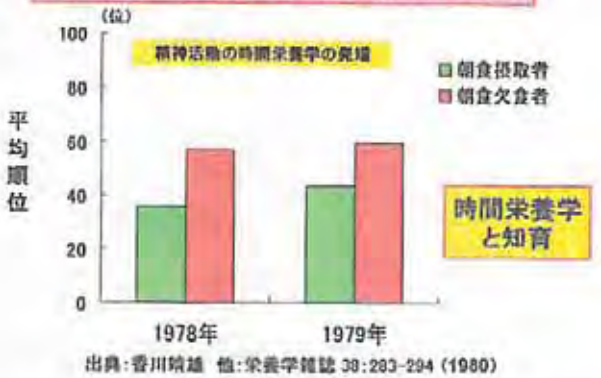
主要時計遺伝子による概日リズムの発振機構



やさしい栄養学174頁　時間栄養学（2009）

12

朝食を摂取した学生は学業成績が高い
全寮制、同一授業、同一食堂の自治医大生を対象



時間栄養学と知育

出典：香川靖雄　他：栄養学雑誌 38:283-294 (1980)

13

朝食をとるかどうかと各教科の正答率

中学校

時間栄養学と知育

朝食をとるか	平均正答率(%) 国語		社会		数学		理科		英語	
必ずとる	81.5	80.5	73.3	71.9	67.7	68.1	69.6	68.3	75.4	73.9
たいていとる	76.8		66.4		60.0		63.1		68.1	
とらないことが多い	73.5	73.5	62.2	61.7	54.8	54.1	59.0	58.9	63.1	62.9
とらない	71.9		60.9		53.0		58.5		61.1	

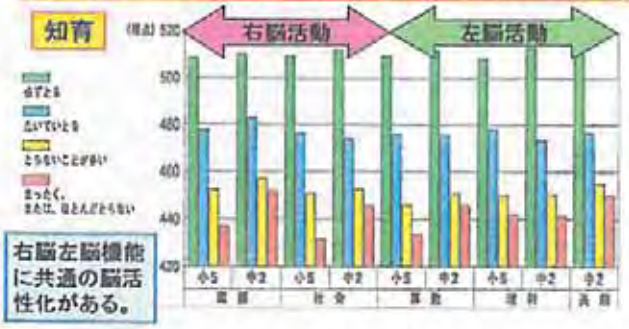
東京都教育委員会ホームページより

14

朝食摂取と成績の相関。全国学力試験。国立教育科学研究所調査（文部科学省、平成16年）

国際的な食と学業成績の論文集はJ Am Diet Assoc 105: 743-760 (2005)

知育

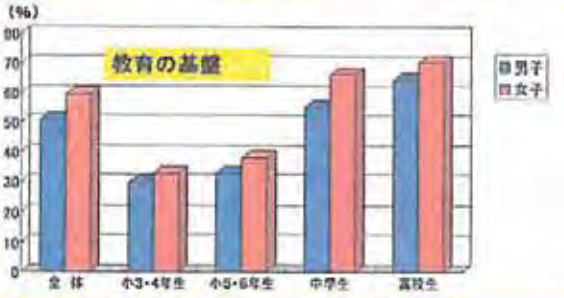


右脳左脳機能に共通の脳活性化がある。

15

日本の青少年が睡眠不足を感じている割合

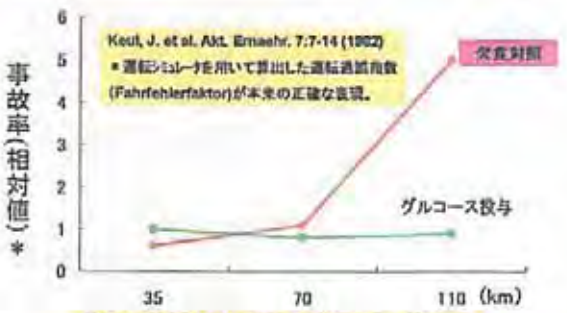
（日本学校保健会：平成16年度児童・生徒の健康状態サーベイランス事業報告）



授業中よく寝る高校生：日本26%、米国11%、中国7%　日本青少年研究所

16

朝食は脳を活性化して交通事故を防ぐ



朝食を摂らないと交通事故を起こしやすい。
飲酒と同様に朝食欠食も処罰の対象？

17

金沢発ディズニーランドへの深夜9時間の運転安全の時間栄養学

1. 昼間8時間を真っ暗な部屋で完全睡眠
2. 深夜に3食摂取
3. 昼間色眼鏡着用
4. メラトニンで昼睡眠

つまり米国人の昼夜と同じにする。

18

栄養素のバランスが肝時計遺伝子の位相同調に必要
Hirao A et al.; PLos One 4:e6909 (2009)

19

食事バランスガイドが勧める朝食が正しく、時計遺伝子の針を朝に合わせて心身の活力が高まる。
http://www.mhlw.go.jp/bunya/kenkou/pdf/eiyou-syokuji2.pdf

食事	主食	副菜	主菜	牛乳・乳製品	果物
朝食	白飯小2杯 2	ひじきの煮物 1	目玉焼き 1		みかん1個 1
昼食	白飯小2杯 2	野菜スープ 1 野菜サラダ 1	ハンバーグ1/2 1.5	チーズ1枚 1 ヨーグルト1杯 1	
夕食	白飯小2杯 2	筑前煮 2 ほうれん草お浸し 1	カキ塩焼き1/2 1 冷奴1/2丁 1		りんご小1/2 1
合計	6	6	4.5	2	2

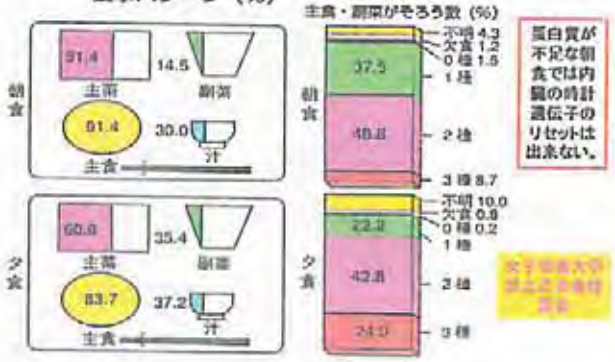
バランスが崩れていないと、適量分を野菜になりにくい

【朝食】 【昼食】 【夕食】

20

朝食を摂ってもその質が問題: 主菜・副菜・汁の揃った朝食は9%

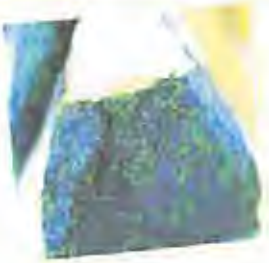
主食・主菜・副菜とその組み合わせからみた食事パターン（%）



21

スマートライフプロジェクトが勧めるお握りだけの朝食が流行していますが、これだけでは時計遺伝子の針は朝に合わせることは出来ません。

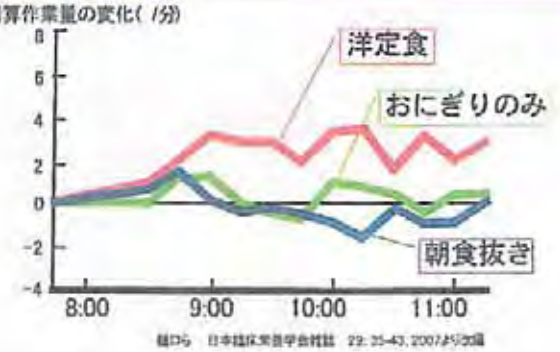
おにぎりでオハヨウ。



22

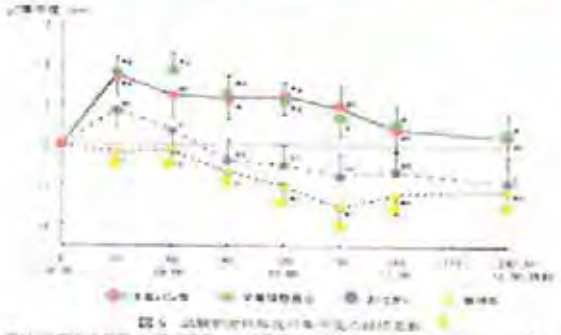
栄養バランスの朝食が脳機能を高める。

暗算作業量の変化（/分）



23

朝食の栄養バランスで集中力が増す。
積口智子 他：日本臨床栄養 29(1) 35-43 (2007)



24

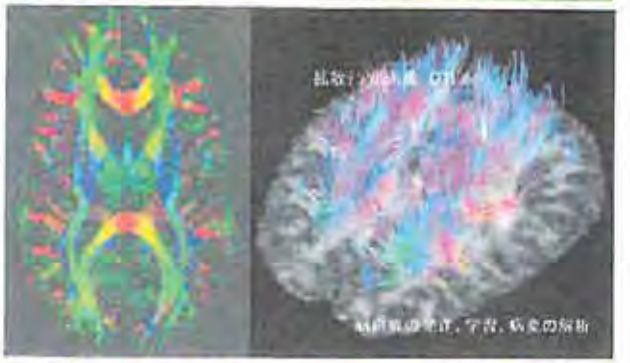
栄養バランスのとれた古来からの朝食でないと時計遺伝子の位相を朝に合せることができない。

栄養に関する遺伝子の人種差

	白人	日本人
エネルギー消費	3000Kcal	2200Kcal
脂肪の燃焼	エネルギー脂肪	筋肉脂肪
牛乳耐性	(+)	(−)
グルコース代謝	強い	弱い
食料文化	牧畜中心	穀物中心

25

教育の成果を練習の跡：神経回路の形成から推定



ピアノが弾けるかなど発達学習、病変は拡散テンソル画像で神経線維の走行で見る。

26

朝食の栄養バランスと認知機能 川島隆太

http://www.fbl.ldac.tohoku.ac.jp/www/docs/100112_asagohan.pdf

	全脳	前頭前野			側頭連合野			側頭連合野
	作業力	記憶力	言語操作	前後思考	空間認知	数的認知	立体認知	図形操作
生活習慣内容	Digit Symbol	数唱順唱	数生成課題	記号課題	認識課題	2D課題	3D課題	マッチ課題
朝食パン食	↓	↓	↓	↓	↓	↓	↓	↓
朝食味噌汁摂取								
朝食ジュース摂取	↓	↓	↓	↓		↓	↓	
朝食紅茶・コーヒー摂取								
朝食日本茶摂取								
朝食野菜摂取								
朝食おかずなし	↓	↓	↓		↓	↓	↓	↓
食事の楽しみの少さ								
朝食をとる	↓	↓	↓	↓	↓	↓	↓	↓
手作り食事割合								
食事中の雑談の会話が多い								

有意な正相関　↓ 有意な負相関

27

四群点数法の基本：肥満の解決法

28

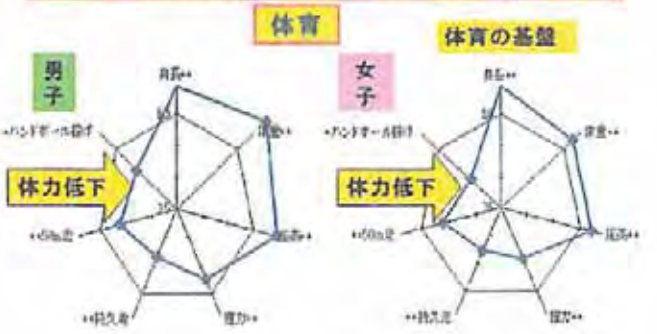
体育においても朝食欠食は有害

朝食摂取状況別20メートルシャトルランの折り返し数

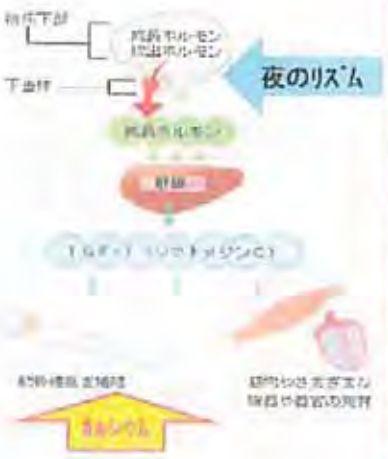
29

15歳高校生の体格測定・体力調査30年前との比較

—昭和47年50% —平成14年 東京都



30

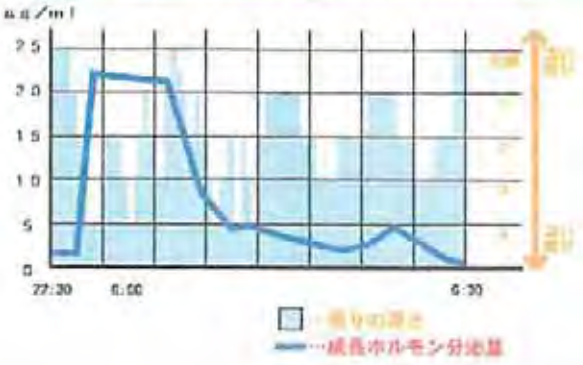


骨粗鬆症予防のCa摂取法

成長ホルモンは就寝初期に分泌されるのでカルシウムもその時期数時間前に摂取する。

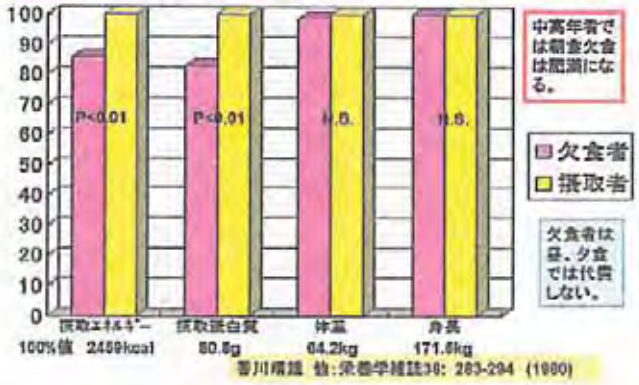
31

寝る子は育つ。成長ホルモンは22時から1時がピークなのでカルシウムは夕食で補給し骨形成



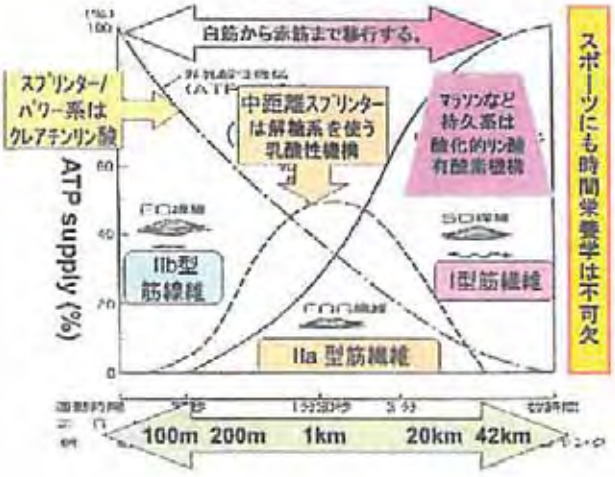
32

若い朝食欠食者は摂取量が摂取者に比して14%少ないが体重は等しい。欠食者は活動量減少 (n=106、男)



香川靖雄 他：栄養学雑誌38: 283-294 (1980)

33



34

栄養指導の良否は実績で判定

箱根の「神」食事で!
女子栄養大学の成果

清井俊平監督の知り合いの管理栄養士さんに1年から栄養管理指導をしてもらっている。

元日の午後1時、箱根駅伝往路の号砲を10時間前に控え、東洋大学の"山の神"柏原竜二選手(20)は神奈川・平塚の宿舎で夕食前のデザートにヨーグルトを食べていた。

2④

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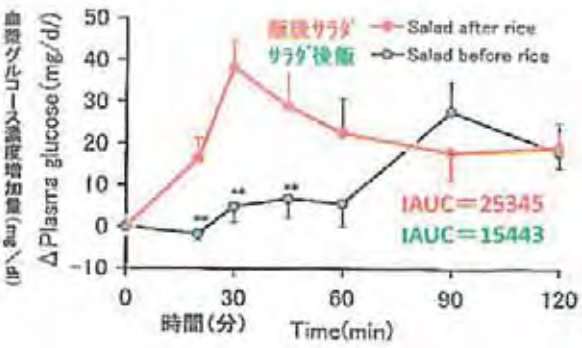


Fig. 1 Incremental plasma glucose response in 10 healthy subjects

金本郁男他. 糖尿病53(2):96-101, 2010

36

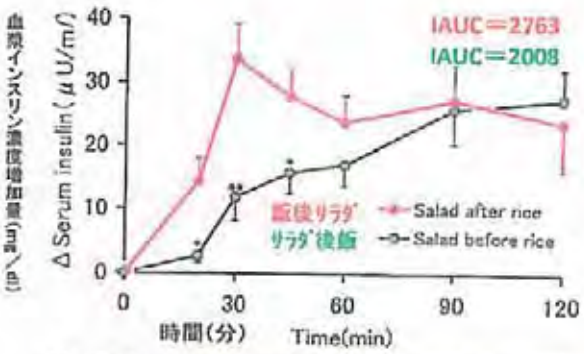
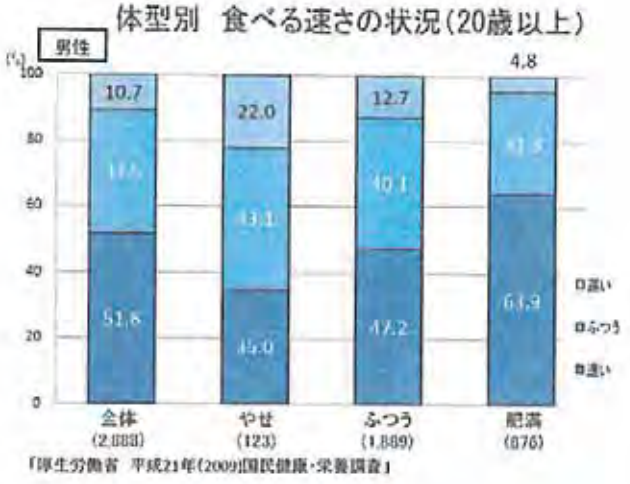


Fig. 2 Incremental serum insulin responses in 10 healthy subjects.

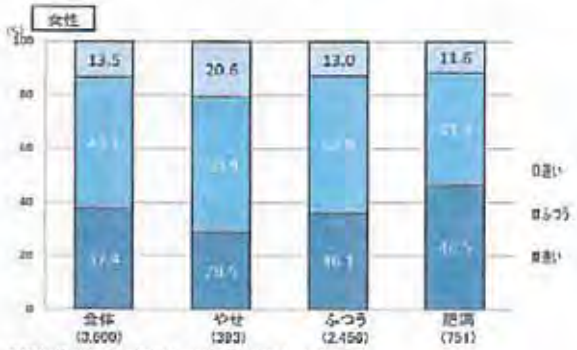
金本郁男他. 糖尿病53(2):96-101, 2010

37



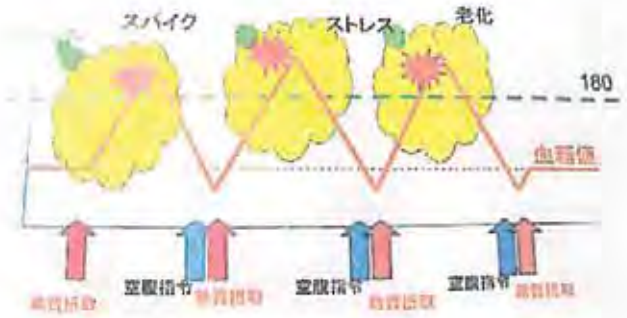
38

体型別 食べる速さの状況(20歳以上)



39

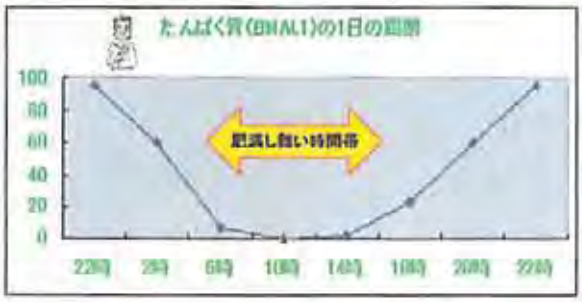
急激な血糖値上昇(180mg/dl)に対しインスリンが分泌されて脂肪を合成する。
ゆっくりと食べ、野菜から食べ、全粒穀類や酢で吸収速度を低下させる。



40

時計遺伝子蛋白質Bmal1の日周リズム

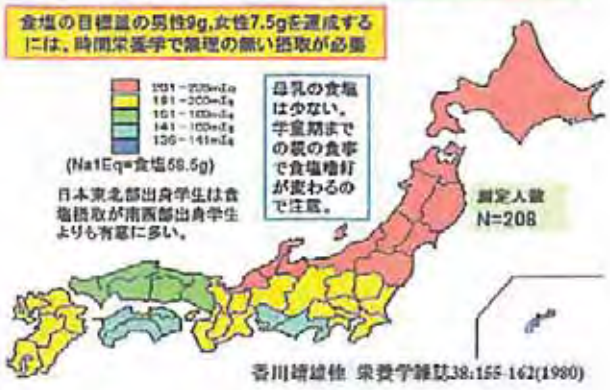
Bmal1は脂肪合成を促進するので夜食で肥満



41

県別Na排出量全寮制自治医大学生(24時間尿)
同一献立の食事でも調味料と汁を摂る量で地域 差

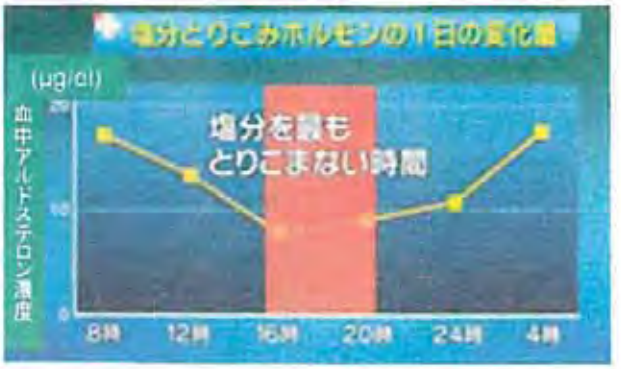
食塩の目標量の男性9g,女性7.5gを達成するには、時間栄養学で無理の無い摂取が必要



香川靖雄他 栄養学雑誌38:155-162(1980)

42

食塩を摂取するなら夕食：長続きする食生活
加藤秀夫 他：*JJPEN*23:557-593(2001)



43

生物にも2種類の時計がある。時計遺伝子のリズムと寿命の回数券テロメア

振り子時計：Oscillatory Clock
日周リズムと時計遺伝子

砂時計：Hourglass Clock
老化とテロメア

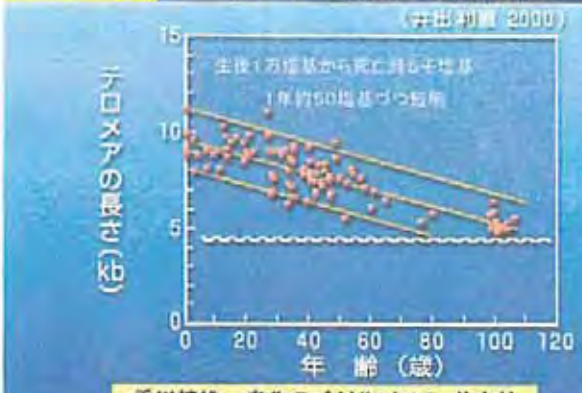
44

細胞分裂時計 テロメアと細胞分裂

45

2009年ノーベル医学生理学賞

テロメアの長さと年齢

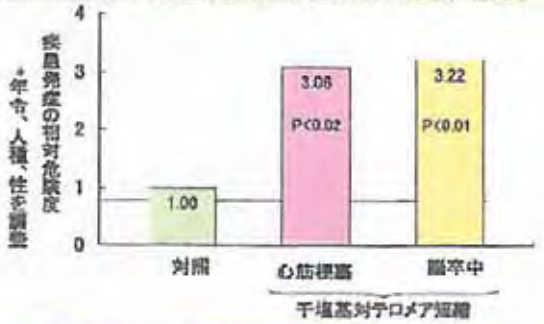


香川靖雄：老化のバイオサイエンス 羊土社

46

7年間でテロメアが千塩基対短くなると心筋梗塞、脳卒中発症が約3倍に増える

Cardiovascular Health Studyのコホート5,201名中の415名、75歳以下

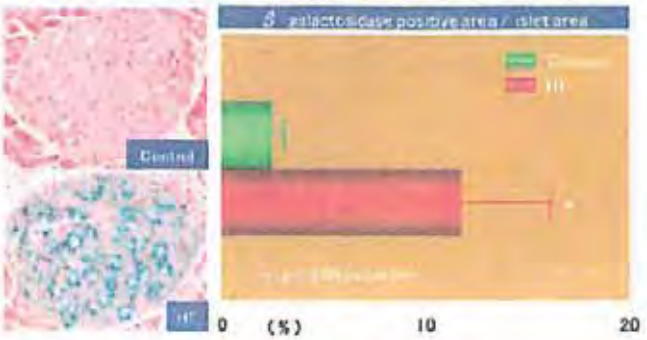


文献：Fitzpatrik AL et al Am. J. Epidemiol 155: 14-21 (2007)

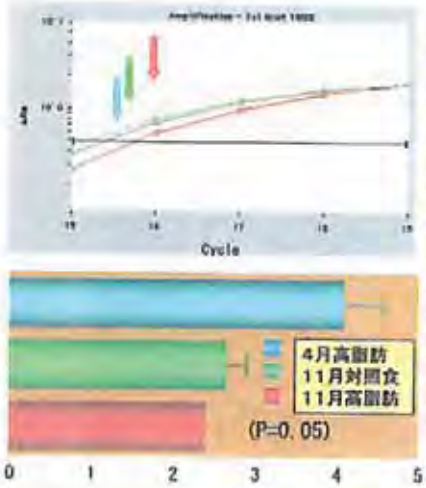
47

高脂質食を11ヶ月投与したマウスはインスリン抵抗性→β細胞分裂促進→分裂能低下、老化マーカー(β-galactosidase)出現→糖尿病

曽根英明、香川靖雄：糖尿病の膵島老化説：Diabetologia 48 (1) 56-67 (2005)



48



糖尿病発症とβ細胞のテロメア短縮の実測値

糖尿病の膵島老化説

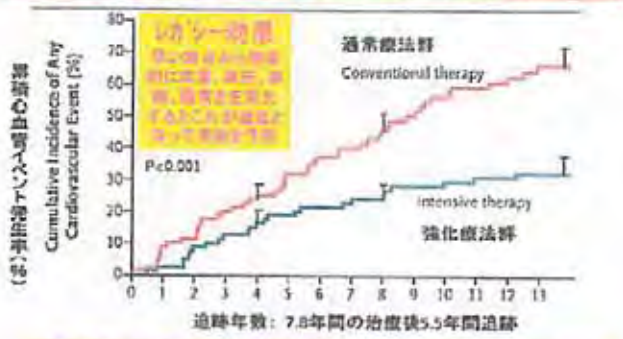
高脂肪食11ヶ月投与による膵臓β-細胞のテロメア長の短縮を増幅系で測定した。

Sone H and Kagawa Y *Diabetologia* 48: 58-67 (2005)

香川靖雄 *Nutr Rev.* 70 (2012)

高脂肪食によるβ細胞増殖促進と早期増殖停止
Division potential and lifespan of mouse pancreatic β-cell fed with high fat diet

通常療法群に比し強化療法群では複合心血管イベント発症率が59%（P<0.001）まで低下



Gade P et al: Effect of a multifactorial intervention on mortality in type 2 diabetes. N Eng J Med 358:580-591, 2008

強化療法群と通常療法群の13年後の心血管イベントの比較

Gade P et al: Effect of a multifactorial intervention on mortality in type 2 diabetes. N Eng J Med 358:580-591, 2008

テロメアを保つ線維、ビタミンE、損傷する肥満度、脂肪、リノール酸
Cassidy A et al. Am J Clin Nutr 91:1273-1280 (2010)

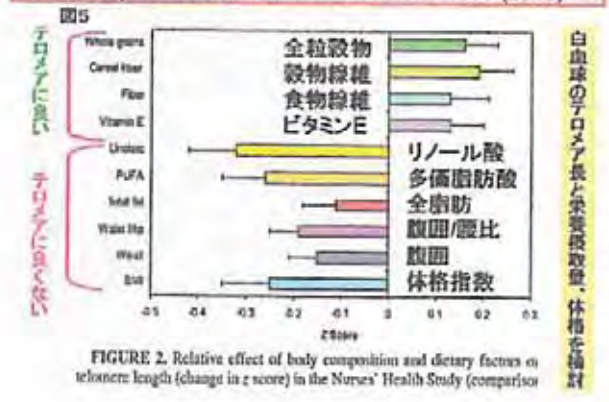
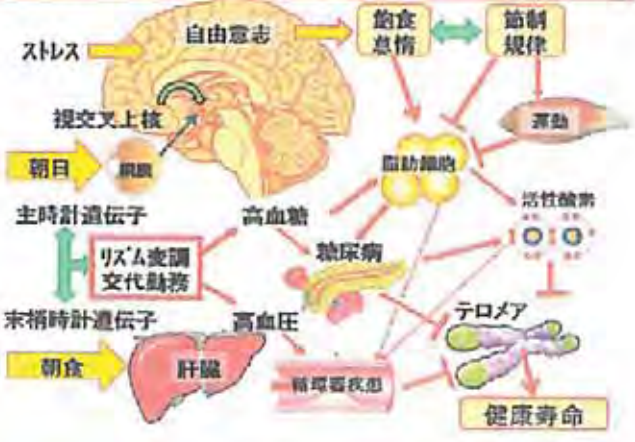
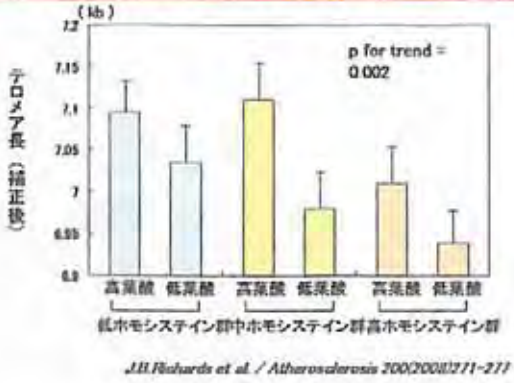


FIGURE 2. Relative effect of body composition and dietary factors on telomere length (change in z score) in the Nurses' Health Study (comparison

時計遺伝子のリズム変調は高血糖、高血圧を介しテロメア寿命へ：香川：Nutr Rev 70 (2012)



葉酸が不足すると有害なホモシステイン濃度が上昇して組織を損傷し寿命の回数券のテロメア長が短縮する。



J.H.Richards et al. / Atherosclerosis 200(2008)371-377

55

世界60カ国で米を含む穀類への法的葉酸強化
Compulsory Folate Fortification of Cereals by 60 Coutries

January 2012: Fortifying with at least iron and/or folic acid (64カ国)

http://www.sph.emory.edu/wheatflour/globalmap.php

56

葉酸+ビタミンB6+ビタミンB12は日本人の虚血性心疾患を予防する。
Ishihara J, Iso H et al.: J Am Coll Nutr. 2008;27(1):127-136.

初回調査時点で循環器病にも癌にもなっていなかった男女約4万人を約11年追跡。
日本人468,472人・年の多目的コホート研究(JPHC研究)

図2 葉酸、ビタミンB6、ビタミンB12摂取量高群と低群の組み合わせと心筋梗塞のリスク

葉酸平均摂取量
高＝486μg
低＝296μg
今の推奨量の約2倍ならば有効

57

2010年日本栄養改善学会講演：葉酸栄養の改善は葉酸米で

ご使用方法

❶「葉酸米」を洗米後のお米（または無洗米）に、炊わずにそのまま加えてください。
❷水加減をし、まぜ合わせ、いつもどおりに炊飯してください。

※写真は小さじ1/2杯、3合分です。スプーンは付いていません。

「葉酸米」0.75g（1合当たりの設定量）に含まれる成分栄養成分含量・保健機能食品基準値に占める割合

栄養素	保健機能食品基準値	含有量（0.75g/1合）	割合	参考
ビタミンB6	1.0mg	1.4	140%	0.00
ビタミンB12	1.0μg	0.5	50%	0.12
ビタミンB6+B12	2.0mg	2.4	120%	0
葉酸	200μg	200	100%	12

「葉酸米」の栄養成分含量
「葉酸米」の保健機能食品基準値に占める割合

58

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10時以降に就寝する子どもの割合（単位%）

	1歳6月	2歳	3歳	4歳	5歳
1980年	25	29	22	13	10
1990年	38	41	36	23	17
2000年	55	59	52	39	40

（日本小児保健協会「幼児健康度調査報告書」より）

60

子供の脳はいかにして人間の脳となるか

① 生後4か月までの時期(昼夜の区別ができるようになる)に活性を増す神経系は、主として前脳辺縁系を発達させ、母子関係・環境順応・記憶機能・大脳左右機能分化に必要
健全な母子関係が人格に必要

② 4か月から幼児期初期(昼寝が2回から1回になる)に活性を増す神経系は、認知機能統合に関与する連合野の発達に必要　　脳の概日リズムの乱れで性格異常

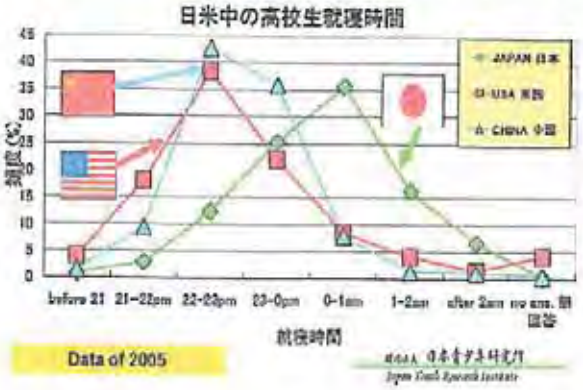
③ 幼児期中後期(昼寝を少なくして昼夜二相性の睡眠になる)に活性を増す神経系は、非運動系大脳基定核を介し、社会性・動機づけの発達に必要 → 反社会行動

瀬川昌也：遺伝.58巻3号,2004年5月号

61

Bedtime of High School Students of Japan, USA and China

日米中の高校生就寝時間



Data of 2005

62

米国は国民の活力向上のため、学校朝食を全州で始めた
学童の健康増進、学業成績の向上、非行減少を毎年詳細に確認。

学校朝食シンポジウムにおけるTim Johnson上院議員の講演
国際競争に打ち勝つために我々は教育された生産的な労働力を持たねばならない。そのためには学童の教育が必要である。
学校朝食は米国の学童の育成と教育の最良の方法の一つである

Remarks to the Symposium on Breakfast and Learning in Children

Senator Tim Johnson
http://www.fns.usda.gov/cnd/Breakfast/Symposium/Johnson.htm

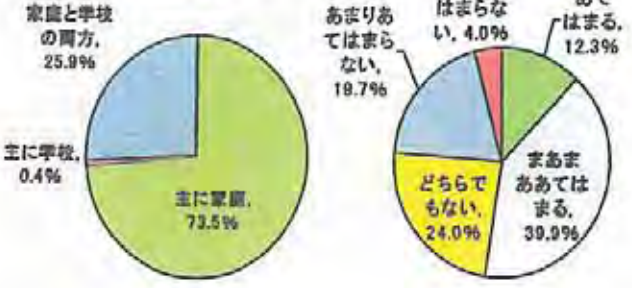
63

朝食欠食習慣化の時期
厚生労働省平成9年国民栄養調査

64

子どものしつけは家庭でが7割。しかし、早寝・早起き・朝ごはんは半分程度しかあてはまらない

Q子どものしつけは家庭と学校どちらで行うべきか
(単一回答、n=1235)



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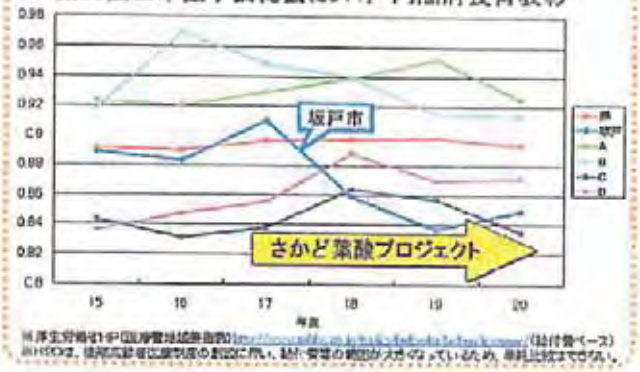
厚生省国民栄養調査、厚生労働省国民健康・栄養調査より

66

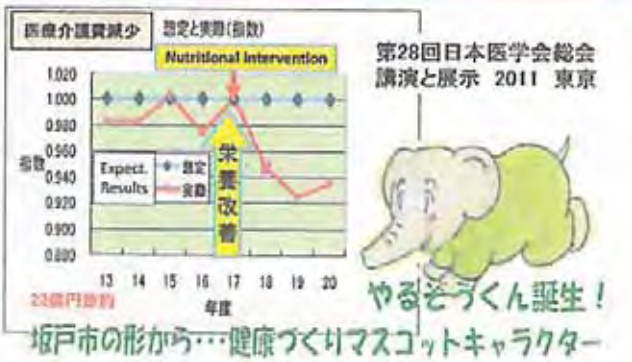
朝ごはん摂取運動

成人式で冊子配布

生活習慣改善、葉酸で心筋梗塞・脳卒中・認知症の激減
（坂戸市民医療費削減10億円　医療費全国の85%）
第28回日本医学会総会(2011)　内閣府食育表彰



時間栄養学を含む「さかど葉酸プロジェクト」の成果



第28回日本医学会総会
講演と展示　2011　東京

やるぞうくん誕生！

坂戸市の形から…健康づくりマスコットキャラクター

Fig1 はスライド 12 に、Fig4 はスライド 54 に、
Fig5 はスライド 48.49 に対応しています。

NUTRITION REVIEWS 70 YEARS 1942-2012

Special Article

From clock genes to telomeres in the regulation of the healthspan

Yasuo Kagawa

Biological clocks are classified into oscillatory (clock genes) and unidirectional hourglass clocks (telomeres). Clock genes align behavioral and biochemical processes with the day/night cycle. Telomeres, the repeated series of DNA sequences that cap the ends of chromosomes, become shorter during cell division. Shortened telomeres have been documented in various pathological states associated with aging. Human activity is driven by NADH and ATP produced from nutrients, and the resulting NAD and AMP play a predominant role in energy regulation. Caloric restriction increases both AMP and NAD and is known to extend the healthspan (healthy lifespan) of animals. Silent information regulator T1 (SIRT1), the NAD-dependent deacetylase, attenuates telomere shortening, while peroxisome proliferator-activated receptor γ coactivator 1α (PGC-1α), a master modulator of gene expression, is phosphorylated by AMP kinase and deacetylated by SIRT1. Thus, PGC-1α is a key component of the circadian oscillator that integrates the mammalian clock and energy metabolism. Reactive oxygen species produced in clock mutants result in telomere shortening. The circadian rhythms produced by clock genes and lifestyle factors are ultimately controlled by the human brain and drive homeostatic and hedonic feeding and daily activity.

INTRODUCTION

The survival of all organisms depends on the dynamic control of energy metabolism in response to environmental changes.[1] Tens of kilograms of adenosine triphosphate (ATP) are synthesized and hydrolyzed in a human body on a daily basis.[1] Endogenous, time-keeping clock genes are necessary to anticipate short-term environmental changes and to initiate internal adjustments of energy demand in advance of the appropriate environmental time.[2] Clock genes coordinate the energy demand of physiological processes with external day/night cycles by driving the rhythmic transcription of many genes.[2,3]

Oscillation of clock genes

Circadian rhythms are produced by an autoregulatory transcriptional and translational negative feedback loop that revolves around a 24-h cycle.[2,3] For the purpose of clarity, only the core feedback loop is described in this review (Figure 1, Table 1).[2] Briefly, the transcriptional regulators, CLOCK and BMAL1, form a heterodimer (CLOCK/BMAL1). This heterodimer binds to E-box and activates the transcription of the *Per* and *Cry* genes. In turn, the product, a PER/CRY complex (negative element), suppresses the transcription of its own genes by blocking CLOCK/BMAL1 activity (Figure 1, upper right). The resulting decrease in PER/CRY subsequently results in the resumption of the transcription of both *Per* and *Cry*. By alternating actions of transcription activators and repressors, this negative feedback loop, with its inherent time delay, acts like an oscillator (Figure 1, upper left).[2–4] In mammals, there are several species-specific subgroups of these components, including PER1–3 among humans, and PER 1–3 in mice. A further dozen candidate genes and proteins, such as protein kinases, have been shown to

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Key words: biological clock, circadian rhythm, lifespan, reactive oxygen species, telomere

doi:10.1111/j.1753-4887.2012.00504.x

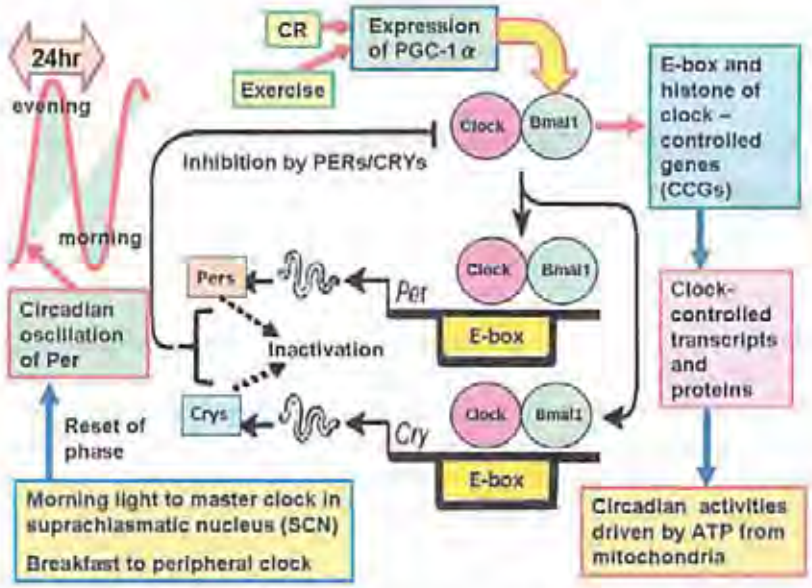

Figure 1 **A simplified negative feedback loop of the mechanism responsible for human circadian rhythm generation.**[2,3] The process begins when Clock/Bmal1 heterodimers drive the transcription of *Per* and *Cry* genes and translate the mRNA into Per and Cry proteins, respectively. When levels of Per and Cry proteins reach a threshold, they form heterodimers and inhibit Clock/Bmal1-mediated transcription of their own genes. The resulting decrease in Per/Cry resumes their own transcription. This feedback loop takes about 25 h, thereby resulting in a free-run circadian rhythm, which is converted into a 24-h diurnal rhythm by resetting the clock genes with morning light and breakfast.
Abbreviations: Bmal1, brain and muscle ARNT-like protein 1; Clock, circadian locomotor output cycles kaput; CR, caloric restriction; Cry/Crys, cryptochrome; E-box, nucleotide sequence CACGTG in the regulatory region of a gene; PGC-1α, peroxisome proliferator-activated receptor coactivator α; Per/Pers, period.

possess additional roles in the circadian gene network[3,4]; for example, expression profiles of all 49 mouse nuclear receptors in tissues revealed that 25 nuclear receptors operate with a rhythmic cycle.[5] The activation and inactivation of all of these protein products regulate the circadian rhythm.[2–4]

While the master clock genes in the suprachiasmatic nucleus (SCN) are reset by light, peripheral clock genes in the liver and intestine are reset by food availability (Figure 1, lower left).[2,3] The hundreds of genes responsible for metabolism, which are regulated by E-box, are all termed clock-controlled genes (CCGs) (Figure 1, lower left) and produce about 10% of all transcripts.[3–5] In addition, CLOCK acetylates histone H3 at CCG promoters to activate transcription in a circadian manner.[3] Simply stated, CCGs couple the circadian clock to divergent metabolic outputs.[3–5]

Regulation of energy metabolism by clock genes

The mammalian clock regulates major aspects of energy metabolism, including mitochondrial ATP synthesis (Figure 2, center).[1] Specifically, clock genes regulate circadian human nutrition and activity by influencing the center of the following metabolic cycle: nutrients→NADH→$\Delta\mu H^+$→ATP→ADP (AMP)→NAD→nutrients, where $\Delta\mu H^+$ is the electrochemical potential of protons across the mitochondrial inner membrane.[1] Energy demand of psychosomatic activity is mainly matched to ATP supply by actions of ATP

Table 1 **The major components of clock genes.**

Gene	Explanation of abbreviation	Protein	Human protein[b]	Roles in transcription
Clock[a]	Circadian locomotor output cycles kaput	clock	CLOCK	positive
Bmal1	Brain and muscle ARNT-like protein 1	bmal1	BMAL1	positive
Per	Period	per	PER	negative
Cry	Cryptochrome	cry	CRY	negative
E-box	*cis*-regulatory element (-CACGTG-)			binding site of CLOCK/BMAL1

[a] When referring to clock genes in general, use lowercase "clock," but use "*Clock*" when specifying the *Clock* gene.
[b] When referring to mice clock proteins, capitalize only the first letter, as in "Clock," "Per," etc.

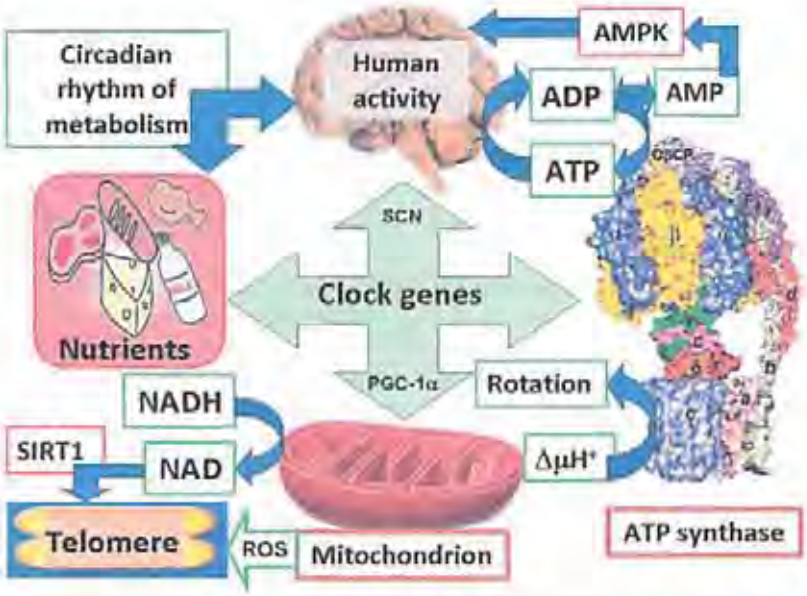

Figure 2 **Central role of clock genes in the regulation of energy metabolism and telomere shortening.** Nutrients reduce NAD, while the electron transport system in the mitochondria drives protons to form $\Delta\mu H^+$, which rotates the central axis of ATP synthase.[1] Clock genes in the suprachiasmatic nucleus (SCN) in the hypothalamus and PGC-1α in the cells regulate activities driven by ATP.[3,4] The resulting AMP activates AMPK.[4] The circadian rhythm of metabolism eventually accumulates NAD,[5] a substrate for SIRT1,[19] which maintains telomere length.[17,18] On the other hand, ROS from mitochondria reduce telomere length.[16–18] *Abbreviations:* AMPK, adenosine monophosphate-activated protein kinase; CCG, clock-controlled genes; $\Delta\mu H^+$, electrochemical potential difference of protons across the membrane; ROS, reactive oxygen species; SIRT1 (or NAD-dependent deacetylase sirtuin-1), silent mating type information regulation 2 homolog 1.

synthase (Figure 2, right). To drive ATP synthase, nutrients are dehydrogenated via the citric acid cycle, which also reduces NAD to NADH (Figure 2, lower left).[1] In turn, the electron transport system uses O_2 to oxidize NADH to NAD and also to pump H^+ against $\Delta\mu H^+$.[1] This H^+ flux induces a conformational change in ATP synthase, which in turn produces ATP from ADP + Pi (Figure 2, right).[1] Consumption of ATP, which increases levels of ADP + Pi, results in instant $\Delta\mu H^+$-driven synthesis of ATP as well as increases in electron transport activity.[1] For purposes of monitoring energy demand, the myokinase reaction (2ADP = ATP + AMP) increases AMP levels when ADP levels become elevated (Figure 2, upper right).[1] Conversely, a sedentary lifestyle lowers the ADP level, which in turn inhibits ATP synthase.[1] The resulting increase in $\Delta\mu H^+$ decreases electron transport activity (respiratory control).[1]

Thus, human activity produces two energy-responsive signaling molecules that play a predominant role in metabolic control: NAD and AMP.[1] Normal cellular physiological functions are regulated by NAD and AMP levels to maintain homeostasis. AMP activates AMP-activated protein kinase (AMPK), which serves as a cellular signal to activate the ATP-synthesizing system and to inhibit the ATP-consuming system. ATP-synthesizing reactions include glycolysis, β-oxidation, and mitochondrial biogenesis, while ATP-consuming ones include glycogen synthesis and fatty-acid synthesis.[1,4] AMPK also phosphorylates and destabilizes the clock component CRY1, which transduces nutrient signals to circadian clocks within peripheral organs.[4] The level of NAD also displays circadian oscillation in cells (one of the metabolites that show circadian rhythm).[3,5] This oscillation occurs because the gene encoding the rate-limiting enzyme for NAD biosynthesis, nicotinamide phosphoribosyltransferase, is a direct target of CLOCK/BMAL1, because of E-box in the promoter.[6] Together, these mechanisms link circadian rhythms with metabolism, entrain the clock genes, and also extend the healthspan (healthy lifespan),[6] defined as lifespan without limitations for health reasons in physical and social activities.

Telomere shortening and energy metabolism

Chromosome telomeres play a key role in regulating the lifespan of mammalian cells[7] and are involved in age-related nutritional diseases, such as obesity, diabetes, and atherosclerosis.[7] The inverse relationship between caloric intake and healthspan[8] suggests a role for nutrient-responsive signaling molecules in the regulation of telomeres by energy metabolism. Telomere DNA comprises noncoding double-stranded repeats of G-rich

tandem DNA sequences (TTAGGG) at the ends of chromosomes.[7] Since replication of eukaryotic linear chromosomes is incomplete and leaves terminal gaps at the chromosome ends, telomeres inevitably shorten with every cell cycle.[7] In other words, the telomere acts as a mitotic clock. After a limited number of divisions, most eukaryotic cells grown in culture will undergo a terminal growth arrest in the G1 phase of the cell cycle, termed "cellular senescence."[7] The senescence phenotype is characterized by altered cellular morphology, decreased levels of Ki-67, the cellular proliferation marker, and increased activity of senescence-associated β-galactosidase (SA-β-GAL).[7] This growth arrest is thought to be a consequence of progressive telomere shortening. In fact, the cyclin-dependent kinase inhibitors p21 and p16 and the regulatory protein p53 have been shown to execute and maintain the cell cycle arrest in senescence.[7] Thus, telomere length is a parameter that records both replicative history and exposure to environmental stress.[7] Conversely, no cellular senescence occurs in cancer cells because telomerase extends most chromosome ends during each S phase, resulting in enough telomere length to support cell activity.[9]

Human fibroblasts obtained from donors of various ages (0–97 years) indicate an age-dependent decline of energy metabolism.[10] For instance, the levels of respiratory and protein synthesis in a 97-year-old donor were only about 15% of those observed in fetal donors.[10] The age-related reduction in mitochondrial function is under the control of telomere length.[11,12] Indeed, both mitochondrial activity and mitochondrial protein synthesis in the cytoplasts (cells devoid of nucleus) of aged donors were completely restored to the fetal level by fusing HeLa ρ^0 cells (cells devoid of mitochondrial DNA) that had sufficient telomere length synthesized by telomerase.[11] A recent study revealed that telomere dysfunction induces mitochondrial compromise in mice null for either telomerase reverse transcriptase or telomerase RNA component genes through profound repression of PGC-1α (peroxisome proliferator-activated receptor γ coactivator 1α, a master modulator of gene expression) as well as the downstream network regulating energy metabolism.[12] The repression of PGC-1α is caused by the binding of p53 on the promoter of PGC-1α, whereas removal of p53 restores the amount of PGC-1α mRNA.[12] Thus, telomere shortening causes age-dependent decline of energy metabolism through the telomere–p53–PGC-1α– mitochondria pathway.

Telomere shortening caused by clock disruption: reactive oxygen species and obesity

Associations between diet, lifestyle factors, and telomere length have attracted the attention of nutritionists interested in extending the healthspan.[8,13] For example, a decrease in waist circumference and body mass index (BMI) has been shown to prevent telomere shortening.[13] Importantly, both of these anthropometric changes can be attained by caloric restriction and exercise.[8] On the other hand, both obesity and stress increase levels of reactive oxygen species (ROS),[14] which are known to shorten telomere length (Figure 2, lower left)[8,15] and oxidize other DNA.[14,15] As will be discussed later, both excessive and deficient electron transport leads to an accumulation of ROS[14] that easily oxidize the GGG triplet within the telomere's DNA.[16] Cell division, which compensates for apoptotic loss of cells caused by obesity-related diseases,[7] inevitably shortens telomere length.[7]

NAD is a substrate for silent information regulator T1 (SIRT1), which acts to deacetylate proteins[3] in order to prevent apoptosis and to indirectly prevent telomere shortening.[17] Moreover, SIRT1 specifically binds to telomeric repeats, as shown by immunoprecipitation assays,[18] and directly protects the telomere from shortening (Figure 2, lower left).[18] The gain of SIRT1 function in *SIRT1*^{wPer} mice led to longer telomeres,[18] while the loss of SIRT1 function in *SIRT1*$^{-/-}$ mice resulted in telomere shortening.[18] Thus, SIRT1 is a positive regulator of telomere length[18] and attenuates telomere shortening[18] associated with aging. For example, the activation of SIRT1 by resveratrol, an antiaging agent, attenuates telomere shortening.[17]

SIRT1 interacts directly with CLOCK and deacetylates BMAL1 and PER2.[3,6] CLOCK/BMAL1 associates with SIRT1 within a chromatin complex that is recruited to CCG promoters in a circadian manner.[3] After binding to E-box, CLOCK acetylates histone H3, PER2 and BMAL1.[3,6] SIRT1 then becomes activated and deacetylates BMAL1, PER2, and histones.[3] Deacetylated PER2 is further phosphorylated[4] and degraded,[4] and a new cycle begins.[6] Thus, SIRT1 constitutes a direct connection between clock machinery and metabolic activity.

Emerging evidence suggests that circadian clocks play a key role in the aging process and that the disruption of the clock results in accelerated aging,[6] which is reflected by telomere shortening.[7] For instance, sleep disturbance causes telomere shortening.[19] *Bmal1* knockout mice as well as cells deficient in BMAL1 show clock disruption, produce a high level of ROS, and experience a reduced lifespan.[20] In unison, these findings[6,7,17,18,20] further illustrate the interrelationship between clock genes and telomere length.

The following sections elucidate how the healthspan is determined by the telomere (hourglass-type biological clock) and how the telomere length is dependent on the function of clock genes (oscillatory biological clocks), which are themselves under the control of brain function.

HEALTHSPAN EXTENSION BY DELAYED EXPRESSION OF RISK ALLELES

If healthspan is determined mainly by risk alleles of life-threatening diseases, such as coronary heart disease, cancer, and type 2 diabetes, the effect of the biological clock on the healthspan may be small. However, recent genome-wide association studies on 1,000,000 polymorphisms revealed that the mere presence of a set of 30 risk alleles does not guarantee a short lifespan among individuals.[21] Indeed, the subjects who survived past 85 years of age were found to carry the same number (26.8 ± 0.11) of disease risk alleles as young controls (26.8 ± 0.10).[21] Thus, the absence of disease loci does not appear to explain the lower morbidity among centenarians.[21] In fact, such results highlight the importance of a healthy lifestyle in preventing the expression of risk alleles[22] and halting telomere shortening.[13]

Centenarian status and avoidance of life-threatening conditions

Centenarians often reach old age by delaying the onset of life-threatening conditions, subsequently experiencing a short duration of morbidity due to major disease at the end of their life.[22] According to Evert et al.,[22] centenarians can be categorized broadly into three morbidity profiles: survivors, delayers, and escapers. Delayers are individuals who did not experience the onset of major diseases until at least the age of 80 years and include approximately 44% of male and 42% of female centenarians.[22] Approximately 32% of male and 15% of female centenarians are escapers, i.e., individuals who attained their 100th year of life without the diagnosis of any major diseases.[22] Finally, the remaining 24% of men and 43% of women who attain the age of 100 years are survivors, or individuals who had a diagnosis of an illness prior to the age of 80 years.[22] Furthermore, approximately 87% of men and 83% of women with prolonged survival delayed or escaped the most lethal diseases of the elderly population,[22] despite the same number of harmful alleles as controls.[21] Thus, the lifestyle of an individual, encompassing caloric restriction,[8,13] regular daily rhythm,[6] and physical activity,[14] may be more relevant to one's healthspan than harmful alleles.

HEALTHSPAN EXTENSION BY CALORIC RESTRICTION

Caloric restriction that maintains adequate nutrition has been shown to delay aging and extend the healthspan in diverse species,[6,8] including rhesus monkeys.[23] By delaying telomere shortening in vitro[7] and in vivo,[13] long-term caloric restriction maintains superior cell survival over a lifetime compared with an ad libitum diet.[8,24] This increase in longevity by caloric restriction is coupled with profound beneficial effects on age-related pathology.[8,23] Specifically, caloric restriction can reduce the incidence of diabetes, cancer, cardiovascular disease, and brain atrophy in primates,[23] a mechanism that evolved to aid in animal survival during famine.[8] Caloric restriction and/or exercise prevents telomere shortening by the mechanisms explained in the following sections. Not surprisingly, significant inverse correlations between telomere length and overweight status,[13] as well as between telomere length and the incidence of cardiovascular disease, have been reported.[25]

Post-translational regulation of PGC-1α by caloric restriction

Caloric restriction and exercise increase both NAD and AMP (Figure 2, lower left and upper right)[1] and activate SIRT1 and AMPK, respectively (Figure 3, lower right and upper left).[26,27] The metabolic actions of SIRT1 are mediated by deacetylation, which uses NAD to produce O-acetyl-ADP-ribose and nicotinamide.[17,18] On the other hand, the metabolic actions of AMPK are mediated by phosphorylation, which relies on AMP acting as an allosteric activator.[4-26] Resveratrol, the powerful antiaging compound,[17] also activates both enzymes.[26] PGC-1α is deacetylated by SIRT1[17,27] and phosphorylated by AMPK (Figure 3, center).[26] The SIRT1/PGC-1α molecular complex can be visualized by coimmunoprecipitation, both in vivo and in vitro.[27] PGC-1α activated by this post-translational modification increases biogenesis of mitochondria and energy consumption (Figure 3, bottom).[26-29] Mitochondria activated by PGC-1α prevent obesity via increased β-oxidation.[28] Thus, obesity-related diseases and the resulting compensatory cell division are prevented by PGC-1α and eventually retard telomere shortening (Figure 3, lower right). The increased NAD seen during caloric restriction (Figure 3, CR) also conserves telomere lengths through specific binding of SIRT1 to telomeres (Figure 3, upper right).[18]

Expression of PGC-1α and PGC-1β by caloric restriction

The post-translational activation of PGC-1α and the gene expression of PGC-1α can both be enhanced by caloric restriction and exercise.[28] In response to caloric restriction, PGC-1α mRNA levels become elevated in the liver in order to increase gluconeogenesis, whereas they rapidly increase in skeletal muscle in response to physical activity.[28] Gene expression of PGC-1β, on the other hand, is regulated by fatty acids.[28] SIRT3 is localized to mitochondria and its expression is increased during caloric restriction and exercise.[29] By expressing several compo-

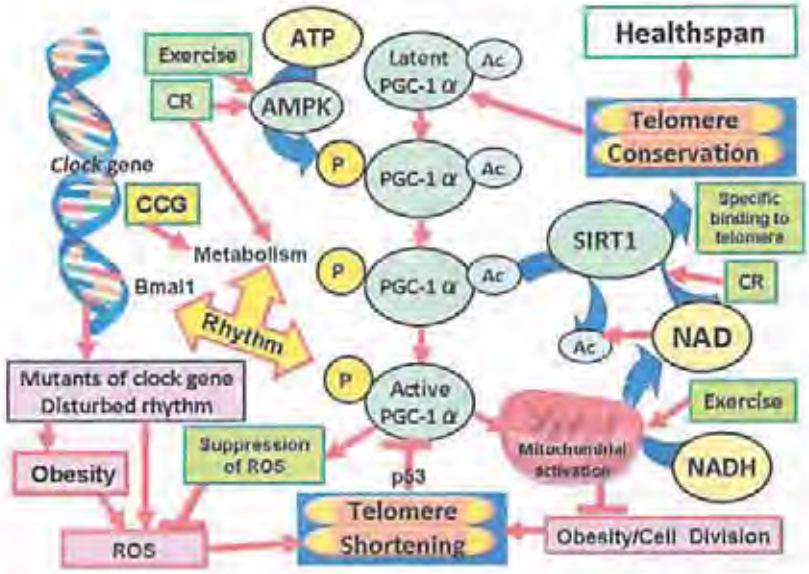

Figure 3 **Relationship between clock gene, telomere, and PGC-1α.** Both caloric restriction (CR) and exercise increase AMP and NAD. PGC-1α is phosphorylated by AMPK and deacetylated by SIRT1, and it activates the mitochondria. Mitochondrial activation increases NAD and protects telomere length by NAD-dependent deacetylation of SIRT1. *Abbreviations*: Ac, acetyl group; CCG, clock-controlled genes; CR, caloric restriction; PGC-1α, peroxisome proliferator-activated receptor coactivator α; ROS, reactive oxygen species.

nents of the respiratory chain, including ATP synthase 5c and cytochrome *c*, the PGC-1α/SIRT3 system acts as a powerful stimulator of mitochondrial biogenesis.[30] In fact, these effects of PGC-1α are lost in SIRT3 knockout mice.[30]

Observations in calorie-restricted animals support the notion that DNA damage caused by ROS may play a causal role in the aging process.[8,15,31] The caloric-restriction-induced reductions in oxidative damage have been attributed to a decline in the rate of ROS generation.[14,31] Caloric restriction increases PGC-1α, which induces ROS-detoxifying enzymes, including glutathione peroxidase-1 and superoxide dismutase 2,[30] and then suppresses ROS production (Figure 3 lower left). Since telomere shortening is caused by both ROS[16] and obesity,[13] prevention of both oxidative stress and obesity explains the antiaging effects of SIRT1/PGC-1α (Figure 3, bottom).[27–31]

HEALTHSPAN EXTENSION BY REGULAR RHYTHM

A regular circadian clock rhythm is another factor that can lead to increased healthspan, while clock disruption is associated with accelerated aging and morbidity.[6,20] Intermittent feeding, which allows the food to be available ad libitum every other day, has been shown to increase healthspan.[8] Additionally, sleep duration is associated with both telomere shortening[18] and mortality.[32] Specifically, research on 1.1 million men and women with ages ranging from 30 years to 102 years revealed that optimal survival is found among those who sleep 7 h per night.[32] Individuals who reported more than 8.5 h or less than 4.5 h of sleep had a 15% greater risk of early death.[32] The major cause of death in this study was cardiovascular diseases, accounting for 42% and 33% of deaths among men and women, respectively. Individuals who slept 7 h per night also had the lowest average BMI levels.[32] In fact, sleep duration has been found to correlate with an altered energy balance.[33] The association between sleep duration and obesity are shown to be U-shaped, with both short and long sleep duration being associated with obesity,[13] which is inversely correlated with telomere length.[13]

Clock gene mutants, obesity, and lifespan

A whole genome scan, which was aimed at identifying genomic regions that may contain quantitative-trait loci for obesity, showed that the *Clock* gene region of chromosome (4q12) may be linked to obesity.[34] In fact, *Clock* polymorphism and the related haplotype of rs1554483G and rs4864548A were associated with a 1.8-fold risk of obesity.[35] *Clock* 3′-UTR rs3749474T was also associated with high energy intake ($P = 0.013$) and sleep factors in obese subjects.[36] The expression of *Clock* in adipose tissue was associated with various parameters of the metabolic

syndrome.[37] Specifically, *Per2* expression level in the visceral adipose tissue depot was inversely correlated with waist circumference,[37] while the three *Clock* studied were negatively correlated with low-density lipoprotein (LDL) cholesterol.[37]

A more direct experiment on homozygous *Clock*-mutant mice showed a greatly attenuated diurnal feeding rhythm, hyperlipidemia, and hyperglycemia.[38] *Bmal1*$^{-/-}$ mice have a reduced average lifespan in comparison with wild-type mice (37.0 ± 12.1 versus approximately 100 weeks) and display symptoms of premature aging.[20] As a potential mechanism for changes in whole-body rhythm, the expression of transcripts encoding selected hypothalamic peptides associated with energy balance was attenuated in *Clock*-mutant mice.[38] In addition, αMUPA mice that overproduce murine urokinase-type plasminogen activator in brain exhibit a higher amplitude in the circadian expression of several clock genes in the liver compared with wild-type mice.[6] In accordance with their robust circadian rhythms, the αMUPA mice never become obese and show spontaneously reduced eating and an increased healthspan.[6] When resveratrol was added to Rat-1 cells, the expression of *Bmal1*, *Per1*, and *Per2* was increased to levels found in αMUPA mice.[39] These results suggest that the circadian clock gene network plays an important role in mammalian energy balance.

Meal frequency, obesity, and healthspan

The disturbance of regular meal frequency and other daily behaviors via prolonged light exposure has been shown to result in a significantly shortened lifespan (658 ± 22.8 versus 844 ± 33.6 days) in young female rats.[40] In a randomized crossover trial of healthy obese women, irregular meal frequency was associated with higher energy intake, smaller postprandial thermogenesis, and higher fasting total and LDL cholesterol,[41] and these may shorten healthspan. Microarray analysis revealed that many periodically expressed clock genes (*Bmal1*, *Per1–3*, and *Cry1–2*) and CCGs in adipose tissues are strongly affected by temporal restricted feeding,[42] which causes tendency toward obesity with a coordinated phase-shift in circadian expression of both oscillator genes and CCGs.[42] The mean lifespan of the rats provided feed every other day[6] represented an 83% increase over that of the ad libitum group,[43] an increase that is larger than that typically observed after caloric restriction (47–60%).[8]

These data support the notion that the oscillatory clock (clock gene) may transfer information to the hourglass clock (telomere) through some molecular mechanism linking both clocks.

MOLECULAR MECHANISM LINKING CLOCK GENE TO TELOMERE

Sensing external signals to clock-controlled genes

The molecular mechanism by which signals from nutritional and other external factors are transferred through the *Clock* gene to telomere length consists of several steps involving the sensing system[28] and CCGs (Figure 3).[3,5] As discussed above, PGC-1α expression is highly responsive to nutritional status, exercise, and other physiological signals.[28] Mice lacking PGC-1α show abnormal diurnal rhythms of activity, body temperature, and metabolic rate, a disruption that is correlated with aberrant expression of clock genes and those involved in energy metabolism.[28] Additionally, analyses of PGC-1α-deficient fibroblasts and mice with liver-specific knockdown of PGC-1α indicate that PGC-1α is required for the maintenance of a cell-autonomous clock-dependent rhythm.[28]

Glucose deprivation from the culture medium activates AMPK and reduces CRY1 stability in the wild-type mouse cells, leading to de-repression of CLOCK/BMAL1 targets.[4] Glucose deprivation did not affect CRY1 of cells lacking AMPK (AMPK$^{-/-}$ cells), which indicates that these effects of glucose limitation are mediated by AMPK.[4] Phosphorylation and destabilization of the clock component CRY1 by AMPK enables CRY1 to transduce nutrient signals to circadian clocks.[4] As shown in Figure 2, both AMP and NAD are increased by caloric restriction and exercise, while AMPK and SIRT1 modify PGC-1α post-translationally (Figure 3, center).[26–28]

Mechanism of oxidative stress and ROS production

Deficiency of BMAL1 leads to premature aging and increased levels of ROS in several tissues of mice.[20,40] ROS cause oxidative damage to proteins, lipids, and DNA, thereby contributing to the development of age-related chronic diseases.[14,15,31] The GGG-triplets in the telomere DNA sequence are a favorable target of ROS, where the formation of 8-oxodG (8-oxo-7,8-dihydro-2'-deoxyguanosine) participates in the acceleration of telomere shortening.[16] The effects of ROS on lifespan were eloquently elucidated by the administration of the antioxidant N-acetyl-L-cysteine to BMAL1-deficient mice.[44] In this study, the removal of ROS significantly increased average and maximal lifespans,[44] perhaps by preventing oxidation of telomere GGG triplets by ROS.[16]

Since genes for enzymes that remove ROS have E-box,[44] their activity has circadian rhythm. The activity of glutathione peroxidase, e.g., follows the rhythm of melatonin, an antioxidant that is secreted nocturnally from the pineal body.[45] Excess food intake accompanied by limited physical activity results in both increased

NADH and decreased ADP.[1] In the absence of ADP, ATP synthase is blocked, thereby resulting in an elevated $\Delta\mu H^+$ (Figure 2, right).[1] High $\Delta\mu H^+$ blocks the electron transport system, and the resulting accumulation of semiquinones of coenzyme Q and flavins interact with molecular oxygen to produce a superoxide anion, a member of ROS.[14,15] In fact, ρ^0 cells devoid of electron transport[10,11] survive in the absence of antioxidants, even under conditions of high O_2.[46] Since both hyperglycemia[46,47] and obesity[48] produce more ROS, patients with short telomeres (less than 80%) are often characterized by a higher BMI, higher 8-oxodG levels, and greater insulin resistance than those with long telomeres.[47] Adipose tissue of obese individuals is characterized by infiltration of macrophages, a site of inflammation-dependent ROS production.[48] A proliferation marker (Ki-67) detected by immunohistochemistry demonstrated that liver tissues of patients with short telomeres were significantly less proliferative than those of patients with longer telomeres.[47] Moreover, obesity caused by clock gene dysregulation produces ROS in the adipose tissue.[48]

The molecular mechanisms of oxidative stress[14,15] caused by circadian disruption[44] and obesity,[47,48] and telomere shortening by ROS[16] offer a logical explanation for the coupling of a peripheral circadian clock to divergent metabolic outputs that lead to telomere shortening.[7,13]

ROS removal and obesity prevention by PGC-1α: healthspan extension

By binding to the *Bmal1* promoter together with retinol-related orphan receptor (RORα), PGC-1α activates transcription of *Bmal1* and induces ROS-detoxifying enzymes.[28,30] Although expression of PGC-1α is elevated in the absence of PGC-1β in all of the tissues, this compensatory increase is not sufficient to prevent metabolic defects, including ROS removal, in PGC-1β knockout mice.[49] PGC-1α decreases ROS[30,50-52] under the influence of SIRT proteins[50] and prevents obesity by increasing mitochondrial biogenesis.[30] The mammalian SIRT family consists of SIRT1 to SIRT7, with SIRT3, SIRT4, and SIRT5 being localized to the mitochondria.[17] SIRT1 directly[18] and indirectly[17] prevents telomere shortening (Figure 3, upper right and bottom). For instance, SIRT1 indirectly reduces ROS through deacetylation of forkhead box O3 (FOXO3), a transcriptional regulator that upregulates catalase and superoxide dismutase.[17]

Considering the post-translational modification of PGC-1α by AMPK and SIRT1 (Figure 3, center)[27,50-52] and the transcriptional regulation of PGC-1α,[28,50-52] PGC-1α is a key component of the circadian oscillator[28,52] that integrates the mammalian clock and energy metabolism and eventually prevents telomere shortening by reducing ROS and BMI (Figure 3). An abundance of indirect and direct evidence[50-53] supports a role for PGC-1α in antiaging and healthspan extension.

TELOMERE SHORTENING IN AGE-RELATED NUTRITIONAL DISEASES

Environmental signals, including stress, light, and breakfast, are received and processed by the cortex, SCN, and liver, respectively (Figure 4).[2] Based on the circadian rhythm of the master and peripheral clocks, voluntary will triggers a hedonic or moderate lifestyle. Visceral obesity caused by the hedonic and irregular[41] lifestyle induces metabolic syndrome accompanied by hypertension and hyperglycemia (Figure 4, center). Telomere shortening has been demonstrated in leukocytes from patients with coronary heart disease,[25,53] hypertension,[54] and diabetes mellitus (Figure 4, lower right).[53] The telomere length of human fibroblasts and lymphocytes shortens by 30–150 base pairs per replication cycle.[7] Because of compensatory cell division that occurs in order to maintain organ-specific function, the rate of telomere shortening of an organ depends largely on the presence of disease.[7,53] Although leukocytes were selected for telomere length measurement primarily because of the availability of stored blood,[25] telomere length is partially synchronized across tissues.[55] Persons endowed with relatively long (or short) telomeres in one type of tissue exhibit relatively long (or short) telomeres in other tissues, regardless of the different proliferative rates of the different tissues.[55] Thus, while the above results may be generalized only to leukocyte telomeres, those from other tissues should be similarly affected.[55] However, when certain tissues are specifically affected and frequent apoptosis is compensated for by cell division, tissue-specific telomere shortening takes place. Examples of tissue-specific telomere shortening include the endothelial cells in hypertension,[25,53] β cells in diabetes,[56,57] and hepatocytes in nonalcoholic fatty liver disease.[47]

Emerging evidence also suggests that alterations in circadian rhythm participate in the pathogenesis of age-related nutritional diseases that include cardiovascular disease and metabolic syndrome.[41,58] Perturbations of the clock gene system, such as those caused by shift work,[59] constitute risk factors for obesity, diabetes mellitus, and cardiovascular disease (Figure 4, lower left).[58,59] For example, in one clock gene study, adults underwent a 10-day laboratory protocol wherein they ate and slept at all phases of the circadian cycle by living on a recurring 28-h day schedule.[59] Subjects ate four isocaloric meals during each 28-h "day." Interestingly, after 10 days on this protocol, circadian misalignment resulted in prediabetes and hypertension in the subjects.[59]

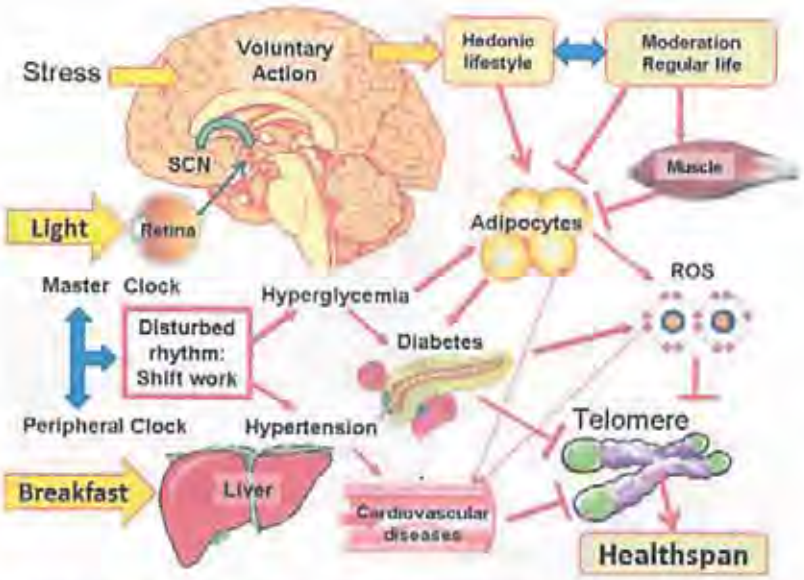

Figure 4 **Central roles played by the brain in controlling clock genes and telomere.** Environmental signals (yellow arrows), including stress, light, and breakfast, are received and processed by the cortex, SCN, and liver, respectively.[2] Disturbed rhythm, such as shift work, causes hypertension and hyperglycemia. Based on the circadian rhythm of the master and peripheral clocks, voluntary will triggers a hedonic or moderate lifestyle. Regular life and exercise prevent obesity-related cardiovascular diseases and type 2 diabetes. The telomere length is shortened by ROS and cell proliferation to compensate cells lost to these diseases. *Abbreviations*: ROS, reactive oxygen species; SCN, suprachiasmatic nucleus.

Diabetes

The telomere length of replicating β cells is decreased in type 2 diabetes.[56] Significant inverse associations were found between telomere length and diabetes as well as between telomere length and fasting insulin level.[23] In vivo, an irregular meal frequency increased insulin resistance,[51] and circadian misalignment caused prediabetes.[59] In vitro, human β-cell-enriched islet cultures can be stimulated to proliferate, but their expansion is limited by growth arrest after 10–15 cell divisions.[56] Analyses of the expression of telomere lengths and SA-β-GAL indicate that cellular senescence is responsible for limiting the number of cell divisions.[56] Compensatory proliferation of β cells caused by insulin resistance of mice fed a high-fat diet occurs during the pathogenesis of type 2 diabetes.[57] As β cells have a limited replication potential, this compensatory proliferation might accelerate cellular senescence and lead to diabetes (Figure 5A,B).[57] After 4 and 11 months on the high-fat diet, measures of telomere length, glucose tolerance tests, and histochemical analyses of Ki-67, p38, SA-β-GAL, and β-cell mass were performed.[57] At 4 months, to compensate for the increased insulin demand, the area under the curve (AUC) for plasma insulin levels during glucose tolerance tests (insulin AUC) was higher, β-cell mass was 3.1-fold greater, and the proliferation of β cells was 2.2-fold higher in the high-fat group versus the control diet group.[57] However, at 11 months, the insulin AUC in the high-fat diet group declined, while the frequency of Ki-67-positive β cells decreased to one-third the level of the control group, and the SA-β-GAL-positive area increased to 4.7-fold the level of the control group (Figure 5A,B).[57] Using the Cawthon method,[60] significant telomere shortening was also demonstrated in the β cells, but not in the brain, of the same mouse fed with a high-fat diet for 11 months compared with mice on a control diet and those fed a high-fat diet for 4 months (Figure 5C,D) (Yanagisawa and Kagawa, Nion Rinsho 2012;70(7):1233–1240). Moreover, small amounts of p38, which is induced by ROS, were found in the β cells of the high-fat diet group but not in those of the control group.[57] These findings suggest that cellular senescence contributes to the pathogenesis of diet-induced diabetes.[57] Furthermore, insulin resistance has been shown to be closely related to β-cell senescence that decreases both ATP and $\Delta\mu H^+$ (Figure 2).[46] In this case, the pancreatic ρ^0 cells fail to secrete insulin because of the ATP deficiency.[46] The hyperglycemia of diabetes causes glycation of proteins, ROS-mediated apoptosis, and telomere shortening in both the β cells and the vascular cells involved in diabetic complications.[46]

Cardiovascular diseases

The telomere length of somatic cells is associated with cardiovascular disease.[7,25,53] Hypertensive subjects exhib-

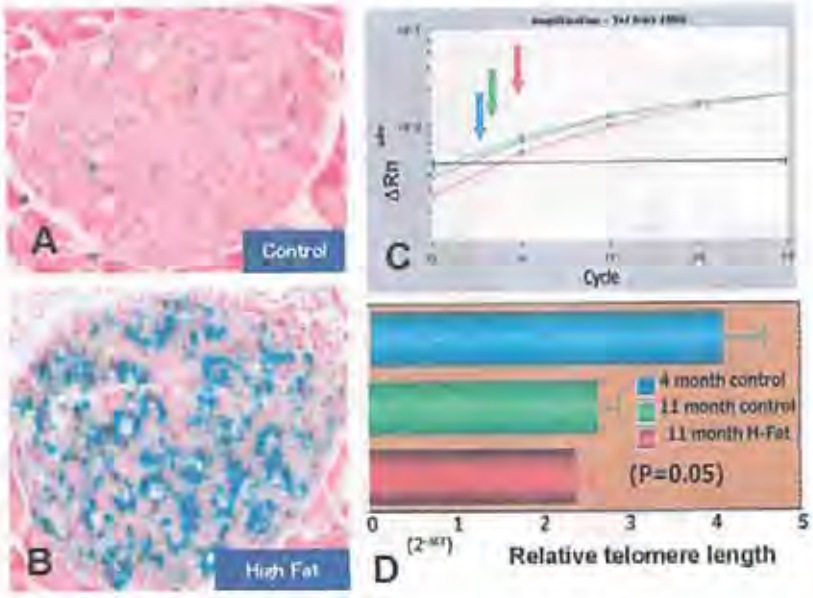

Figure 5. **Detection of β-cell senescence by β-galactosidase (panels A and B) and telomere shortening (panels C and D) induced by high-fat diet.** Pancreatic islet of a mouse fed a control diet (A) or a high-fat diet (B) for 11 months.[57] Blue color indicates β-galactosidase activity.[7,57] Panel C: quantitative polymerase chain reaction (PCR) of telomere. Arrows indicate the points at which the threshold line was crossed (blue: 4-month control, green: 11-month control of the mouse in panel A, and red: 11-month high-fat diet of the mouse in panel B). Telomere length was determined by real-time kinetic quantitative PCR, as reported by Cawthon.[60] *Abbreviations:* Cycle, cycle number of PCR; ΔRn, signal intensity under the specified PCR condition; Ct, threshold cycle value, or the fractional cycle number at which the well's accumulating fluorescence crossed a set threshold that was several standard deviations above baseline fluorescence; T/S ratio, the relative ratio of telomere to single-copy gene, approximated by $[2^{-\Delta Ct(\text{telomere})}/2^{-\Delta Ct(36B4)}]^{-1} = 2^{-\Delta\Delta Ct}$, which is a specific standard primer.

ited shorter age-adjusted telomere length compared with their normotensive peers.[53] Each shortened telomere length corresponded with a threefold increased risk of myocardial infarction and stroke.[23] Additionally, inverse correlations between telomere length, hypertension, and oxidative stress have also been reported.[25,53] Obesity induces ROS production[58] and resulted in disturbed mitochondrial functions in the cardiomyocytes of patients.[61] These results support the hypotheses that telomere shortening may be caused by mechanisms involving ROS. Short telomeres have been detected in senescent endothelial cells and vascular smooth muscle cells from human atherosclerotic plaques[33,34] that contain oxidized LDL. Homocysteine is known to cause caspase-3-dependent apoptosis in cultured human endothelial cells.[62] These apoptotic characteristics were correlated with ROS production following homocysteine treatment.[62] Disturbed mitochondrial function of endothelial cells was associated with signs of increased ROS as well as up to a 30% reduction in telomere length as a result of proapoptotic activation with increased caspase 3/9 activation.[62] Endothelial cell apoptosis caused by hypertension shear stress[63] and hyperhomocysteinemia[62] is compensated for by augmented cell proliferation that results in the telomere shortening.[7,25,53]

Aging in the circadian system: from telomere to clock gene

Aging is associated with changes in the period and amplitude of circadian rhythms,[64,65] combined with a general decline of energy metabolism.[10–12] With advanced age, telomere shortening causes repression of PGC-1α in the control of clock genes.[12] Subsequently, the clock genes regulating circadian function both in vivo and in vitro become disorganized, and the ability of the brain clock to entrain to local time diminishes.[64,65] Circadian expression of clock genes in serum-stimulated senescent human aortic vascular smooth muscle cells was significantly weaker compared with that of young cells.[64] However, introduction of telomerase completely prevented the reduction of clock gene expression associated with senescence,[64] likely due to the restoration of PGC-1α expression.[12] Moreover, when young cells were implanted into young mice or old mice, the implanted cells were effectively entrained by the circadian rhythm of the recipients.[64] Such disruptions in the circadian system appear to accelerate the aging process and contribute to senescence, with some systems being more vulnerable than others.[64,65]

The most pronounced age-related changes in the circadian rhythm of locomotor activity include alterations

in the phase-angle of entrainment to the light-dark cycle,[65] such as those observed among PGC-1α knockout mice.[51,52] In vivo, the circadian clock is controlled by the brain through the SCN.[2] In fact, depletion of brain monoamine levels in young animals can induce changes in the responsiveness of the circadian clock to environmental stimuli that are similar to changes occurring spontaneously in old animals.[65] This suggests that aging alters monoaminergic inputs to the central clock. A vicious cycle of age-dependent circadian clock disorganization and telomere shortening results in accelerated aging of the entire body.

ROLE OF THE BRAIN IN HEALTHSPAN

Both master and peripheral clock genes are ultimately regulated by brain function,[2] while telomere shortening occurs due to obesity-related diseases and oxidative stress resulting from circadian rhythm disturbance (Figure 4).[53] The rhythmic expression of peripheral clock gene products is completely abolished by an SCN lesion in the brain.[2] Telomere shortening is also under the indirect control of the brain through sleeping,[19] feeding,[8,13] and muscle activity.[66] The brain triggers muscle contraction that changes metabolic equivalents from 0.9 to 15 in a normal adult.[1] Through a biochemical cascade, the disturbance of energy balance and rhythm may influence a variety of diseases and lead to telomere shortening (Figure 4). The psychological importance of this homeostatic control system is described in the following section.

Hedonic lifestyle versus moderation: effects on telomere length

The observation that caged animals under caloric restriction using the same chow exhibit a long healthspan as well as telomere length has been confirmed by many experiments.[7,8] On the surface, weight control by maintaining a regular daily rhythm[41] appears simple, but it is difficult because of the complex psychosocial factors involved in initiating and perpetuating healthy habits. Human beings do not simply live in order to prolong their lifespan by following uniform nutritional regimens.

Food is consumed in order to maintain energy balance in response to hormonal regulators of hunger, satiety, and adiposity levels (homeostatic feeding).[66,67] In addition, palatable food stimulates the brain's reward systems independent of their caloric value (hedonic feeding).[67] Such reward-related consumption of high-energy foods can result in caloric intake exceeding requirements and is considered a major culprit of obesity (Figure 4, upper right). Functional magnetic resonance imaging of the brain suggests that the amygdala may be responsive to a general category of biologically relevant stimuli, such as food, whereas separate ventromedial prefrontal systems may be activated on the basis of a perceived reward value of food stimuli.[68]

Interestingly, higher levels of reported dietary restraint by women are associated with shorter leukocyte telomere length, even after adjustment for age and BMI.[69] In this study, the reported dietary restraint was defined as chronic preoccupation with weight and repeated attempts at restricting food intake.[69] The distinction between unhealthy attempts at weight loss, frequently reported by dieters, and successful dietary restraint induced by behavioral intervention is important for interpreting these results.[69] Behaviorally successful dietary restraint decreases regular caloric intake and distress and consequently slows the pace of telomere shortening.[13] Conversely, irregular eating patterns may cause more frequent and greater fluctuations in the metabolome, resulting in accelerated telomere shortening.[70] Those suffering from major depression were found to have a shorter baseline telomere length than those without depression.[71] Chronic stress can lead to overeating and co-elevation of cortisol and insulin.[70] This state of metabolic stress, in turn, promotes abdominal adiposity and, eventually, shorter telomere length (Figure 4, right).[70] Thus, human telomeres are controlled by the brain, just as clock genes are regulated by SCN.

In vitro control of biological clocks and signals from the brain

Although it is easy to demonstrate telomere shortening in cultured cells during cell division[7] and oxidative stress,[16,53] the direct effect of circadian rhythm on the telomere length in vitro is difficult to illustrate.[72] However, by transfecting luciferase into human cells, the in vitro expression of *Bmal1*, *Per1*, *Per2*, and *Cry1* becomes possible to observe in cells from healthy individuals and those with genetically disturbed circadian rhythmicity.[72] Circadian rhythm in vivo is regulated by the central pacemaker SCN, so in vitro control of clock genes needs signals from the brain.[73] These signals include nutrients, metabolites produced by brain-triggered activity, ROS, and hormones. For example, the cortisol analog dexamethasone is widely used to induce circadian gene expression and transiently changes the phase in cultured cells.[73] Thus, in vitro, there are indirect effects of cortisol, nutrients, metabolites, and ROS from tissues on the intracellular telomere maintenance mechanism.[70] Irregular life habits and stress may promote the accumulation of visceral fat and oxidative stress, leading to telomere shortening.

Conscientiousness as a predictor of healthspan

The choice of a lifestyle of hedonism or moderation and weight control depends on the personality and training

status of every individual (Figure 4, upper right). A good lifestyle, including caloric restriction,[8,13] regular daily rhythm,[6,41] and adequate physical activity,[1,14] has a greater impact on healthspan than do harmful alleles.[21] Lifestyle modification is recommended for the prevention of obesity-related diseases. Conscientiousness to observe these guidelines is an important predictor of health and longevity.[74] Conscientiousness (as measured by the NEO Personality Inventory) refers to individual differences in the propensity to follow socially prescribed norms for healthy customs. In fact, conscientiousness is negatively related to the experience of chronic illnesses such as diabetes and stroke.[74] Moreover, lower perceived mental health of patients with chronic heart failure was associated with shorter telomere length.[75] Since adherence to dietary advice is important, the effect of nutritional intervention on clinical data was measured, along with its association with polymorphisms of the serotonin transporter (5-HTTLPR) polymorphism, which is related to personality.[76] Enrolled in the intervention program were 264 Japanese women not taking medication for diabetes, hypercholesterolemia, or hypertension.[76] The SS homozygotes of 5-HTTLPR showed larger decreases in fasting blood glucose in comparison with subjects with other genotypes (SL + LL).[76] The confounding factors were sleep duration (SS > SL + LL) and clock gene polymorphisms (Y. Kagawa et al., unpublished data).

Finally, epidemiological evidence of habit[77,78] supports the close relationship between clock genes (sleep,[19,32] breakfast,[78] and shift work[58,59]) and telomere length[53] that is shortened by ROS[25] and obesity (Figure 4). Oxidative stress and obesity were related to short lifespan in a nutritional and clinical survey of Mongolians.[77] A systematic review of 153 articles revealed a relationship between obesity and skipping breakfast or eating frequency.[78]

CONCLUSION

Advances in the field of chronobiology have revealed the mechanisms governing the circadian rhythm of clock genes (Figures 1 and 4),[2] the central role of clock genes in the regulation of energy metabolism (Figure 2),[1] and the close relationship between telomere length and healthspan (Figure 3). Telomere shortening is accelerated by both ROS and obesity. Genome-wide association studies support the importance of lifestyle in delaying the expression of risk alleles. The molecular mechanisms connecting the daily circadian rhythm of energy metabolism to lifelong telomere shortening involve NAD and AMP, which activate SIRT1 and AMPK, respectively; in addition, PGC-1α plays a central role (Figure 3). Both master and peripheral clock genes are ultimately regulated by brain function. The human brain voluntarily triggers either hedonic or homeostatic behavior and controls daily rhythm, energy metabolism, and, eventually, healthspan (Figure 4). Cardiovascular disease caused by irregular circadian rhythm induces telomere shortening of the endothelial cells. Obesity causes insulin resistance, compensatory β-cell proliferation, and the telomere shortening that eventually results in type 2 diabetes (Figure 5). The elucidation of the long-term effects of circadian rhythm on telomere length will aid the development of optimal personalized nutrition.

Acknowledgements

The author thanks Prof Sadahiko Iwamoto and Dr Kazuhiro Nakayama of Jichi Medical University for their typing of clock gene variants, and Dr YoshikoYanagisawa for her telomere analysis.

Declaration of interest. The author has no relevant interests to declare.

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調 理 実 習

松田 康子　先生
駒場 千佳子先生

家庭科教諭のスキルアップのための調理実習

手軽にできるイタリア料理献立

講師：調理学研究室　准教授　松田康子
（　助教　駒場千佳子）

焼き野菜のマリネ	Sott‘ Olio
ほうれん草ととうもろこしの冷製スープ	Zuppa Fredda ai Spinaci e Granturchi
えびとサフランのリゾット	Risotto allo Zafferano con i Gamberetti
いわしのオーブン焼きトマトソース	Sardine al Forno con la Salsa di Pomodoro
ヨーグルトムース あんずソースがけ	Mousse di Yogurt

焼き野菜のマリネ　（6 人分）　野菜を 1 種ずつ担当し、マリネに漬けて交換

なす		2 本×3 台分	（1 人 2 枚 5mm 厚さ 水に）	1.4.7 の台
かぼちゃ	1/8～1/10 個	120g×3 〃	（1 人 2 枚 5mm 厚さ）	2.5.8 の台
パプリカ（赤）		1/2 個×3〃		3.6.9 の台
サラダ油（なす・かぼちゃ用）				
食塩（野菜の 0.5%塩分）				
＜マリネ液＞	EVオリーブオイル		大さじ 2	
	ニンニクオイル*		小さじ 1/2	
	バルサミコ酢		大さじ 2	
	バジリコ		2 枚（千切り）	

1. なす、かぼちゃは下処理をし、少量のサラダ油で焼いて軽く焦げ目を付け、食塩を振る。マリネ液に漬ける。
2. パプリカは、直火で表面が黒くなるまで焼く。ビニール袋に入れてむらし、皮をむく。食べやすい大きさに切り、食塩を振って、マリネ液に漬ける。

ほうれん草ととうもろこしの冷製スープ　（6 人分）

とうもろこし	1 本半（200g）
玉ねぎ	1/2 個分（120g） 薄切り
ブイヨン*	300+100ml
牛乳	100～150ml
生クリーム（動物性）	50ml
食塩（仕上がりの 0.3%塩分）	小さじ 1/2
ほうれん草のピュレ*	大さじ 3

1. とうもろこしは、実を包丁で切り取り、玉ねぎは薄切りにする。鍋にブイヨン（300ml）と共に入れて、10～15 分煮る。
2. 1 と冷たいブイヨン（100ml）をミキサーにかけ、こす。牛乳、生クリームを加え、食塩で味を整える。
3. ほうれん草のピュレを加える。

えびとサフランのリゾット　（6 人分）

イタリア米	180g	
玉ねぎ	1/4 個(60g)	みじん切り
オリーブオイル	大さじ 1	
白ワイン	大さじ 3	
サフラン	0.2g	
ブイヨン	1 ℓ	
えび(ブラックタイガー)	18 尾	茹で湯、塩(湯の 0.5%塩分)、酢(湯の 3%)
オリーブオイル	大さじ 1	
パルメザンチーズ	大さじ 2	
食塩	小さじ 1/4	
パセリ	適量	みじん切り

1. 白ワインにサフランを入れておく。
2. 鍋にオリーブオイルを温め、玉ねぎを炒める。透き通ったら、米を加えて油がまわったら、サフランのはいった白ワインを加える。ブイヨンを加え、20 分煮る。
3. えびの背腸を取り、塩と酢を入れた湯で 2～3 分茹でる。殻を剥いておく。
4. オリーブオイル、チーズ、食塩で味を整え、えびを加える。盛り付け、パセリをあしらう。

いわしのオーブン焼きトマトソース　（6 人分）

いわし		6 尾　手開き
（食塩 いわしの 0.3%塩分）		今回は使用せず
薄力粉		
サラダ油		大 1
香草パン粉	アンチョビフィレ	6 枚分　みじん切り
	セージ、ローズマリー、イタリアンパセリ	各 3 本　みじん切り
	ニンニクオイル*	大さじ 1/2
	パン粉(乾燥・細)	20g
トマトソース*		180g

1. いわしを手開きする。表面に粉をまぶし、油を温めたフライパンで、表面を焼き付ける。
2. 香草パン粉を合わせる。器にトマトソースを敷く。
3. トマトソースの上に、いわしをのせ、上に香草パン粉をまぶし、230 度のオーブンで 10～15 分焼く。

ヨーグルトムース　あんずソース添え（6人分）

牛乳		100ml
砂糖		40g
板ゼラチン（液体の1～2%）		6g
ヨーグルト（無糖）		250g
生クリーム		100ml
あんずソース（配布）	あんず（缶）	60g
	缶シロップ	15～30ml
	グランマニエ	少々（オレンジのリキュール）
ミントの葉		6枚

1. 板ゼラチンを分量外の水でふやかす。
2. 小鍋に牛乳、砂糖をいれて温め、板ゼラチンを加え溶かす。ヨーグルトに1を加えよく混ぜ、生クリームを加え混ぜ、型に入れて冷やす。
3. あんずソースは、材料をミキサーにかけ、冷やす。固まったヨーグルトゼリーの上に流し、ミントをあしらう(あんず缶は、ヘビーシロップのもの。味をみて砂糖を加えても良い)。

*印の材料の作り方

◇ほうれん草のピュレ(冷蔵庫で3日間ほど保存可能)

ほうれん草	50g
水	10～25g

～フードプロセッサー等で、ピュレ状にする。

◇ニンニクオイル(保存容器に入れて、2週間位保存可能)

にんにく	1玉(みじん切り)
EVオリーブオイル	にんにくの1.5倍～同重量

～忙しいレストランの厨房で手早く使うために用意するもの。みじん切りにしたにんにくに、オイルを注ぐ。

◇トマトソース(冷蔵で1週間、冷凍で1ヶ月保存可能)

ホールトマト缶	1缶約500g
玉ねぎ(みじん切り)	1/4個
にんにく	1かけ
赤唐辛子	1/2本
オリーブオイル	大2
塩・こしょう	

1. 煮込み鍋にオイル、にんにく、赤唐辛子を加え、温めて香りをだす。
2. 玉ねぎを加え、透き通るまで炒めたら、トマト缶を崩しながら炒め、中火で20分程度煮る。

※ブイヨン　ブイヨンキューブ(1個塩分相当量2.3g)を1½に1個の割合で溶かして使用

※オリーブオイル　EV＝エクストラバージン(非加熱用)　　無印＝ピュア(加熱用)

ベテラン家庭科教諭による
授業実践のお話

杉 信子　先生

家庭科教諭レポート①

中高一貫校での家庭科教育、35年の変遷

どこで教える？家庭の技術

私は、中学1年から高校3年までの6年間を指導しています。高校3年生にもなると選択科目の授業でウェディングドレスを作ってファッションショーを企画する生徒も出てきます。その成長ぶりに驚く、中高一貫校の醍醐味を何度も経験してきました。

私が就任した35年前、家庭科は中学1年から3年までで計9単位、高校は計4単位が必修授業でした。現在は中学で計5単位、高校で計2単位とほぼ半減です。普通学校では学力重視なので、技術・芸術分野にさかれる時間は縮少の一途をたどっています。

夏休みの自由作品について、「おばあちゃんに教えてもらいました。お母さんはできないって」などといった生徒の会話を耳にしたり、実際にその技量を見ていたりすると、従来、家庭で伝えられてきた「衣食住」を中心とした家庭内の技術は、家庭よりも学校の中で教える比率が年々高くなっているように思います。

自分の手で物を作り出していく確かで豊かな時間を知ることは、生きていく力の源です。食も然り。まずは健康第一、食べることのコンビニ化は避けたい……と、自分で作ってバランスよく食べる習慣を子どものうちにはぐくむ方法を真剣に問いたいと考えています。

中高教育で足りないのであれば、生涯学習だってよいでしょう。なにがたいせつかを知っている親を育てるのも重要です。

文部科学省に軽視されていると瞹いてばかりはいられません。

「制服の裾を『まつり縫い』で直せる人は手をあげて」――入学したころには1人も手があがらなかったのが、7月にはほとんどの生徒の手があがるようになります。

中学1年の家庭科の授業は「基礎縫い」から。まずは制服を利用して、実践的な力をつけます。

私「表布に糸が見えないように縫えるかな」

生徒「私、不器用なの。できるわけないよ」

私「あら、できてるよ」。ひと針ひと針を見ながら、声をかけていきます。「心をこめてゆっくりね。きっと端まで縫えるからね」。

手縫いとミシンの「基礎縫い」が終わると、9月からは学校で使えるトートバッグの作製。全員の作品を11月の学園祭に展示します。

自分でデザインして縫ったウェディングドレスを着た生徒たちといっしょに。

今月の先生

杉 信子

すぎのぶこ。横浜英和女学院中学高等学校家庭科、情報科教諭。給食室の管理栄養士と組んで食の家庭科教育にも力を入れている。

家庭科教諭レポート⑥

ハードルを高めに…広がる教育の可能性

ウエディングドレスとダイエット

「先生、私はどうしても11月の学園祭までにやせてウエディングドレスを着こなしたいんです。どうしたらよいですか」

毎年のように話題になる、高校3年生の「被服」の授業を選択した生徒の声です。制作テーマは本来「ワンピースドレス」なのですが、「ウエディングドレスにしたい！」という生徒の要望を一部とり入れることに。さらに有志によるウエディングショーの企画案が出てきて、今では学園祭の恒例行事になってしまいました。

100年以上の歴史のある学校の礼拝堂で自作のドレスを着てバージンロードを歩くのですから、生徒は真剣です。この企画をきっかけに、AO入試や公募制推薦入試のプレゼンテーションに臨む生徒も現われるなど、高校3年生の授業は緊張感と意欲にあふれています。

冒頭の質問に、私は次のように答えます。「皆さんならかならず理想的な体重になれるわよ。でも、やつれてはだめ。きれいな体型でつやのよい肌でないと意味がないわね」。そして「ダイエット」の授業を始めます。基本は簡単。「夕食は早めにとり、主食の炭水化物系の食品をとりすぎないこと。主菜、副菜をしっかりとって美しくなりましょう」。さらに一汁三菜の献立と4つの食品群を復習します。

食事の量を変えるには、実際の量を見て食べて現実を知ることがたいせつです（学校では給食のメニューが活用できます）。ちょっと広げて、さまざまなダイエット法やその弊害を話題にするのも一案です。生徒たちは「発表」という現実の目標に向かって楽しそうに、あたりまえにダイエットに成功。私も調子に乗っていっしょに実践したところ、1か月半で3kg減。ちょっとやりすぎました。

家庭科教育の〝生きた学習法〟とは、机上の作業だけでは終わらせないことです。できるだけ実生活、実社会からのさまざまな刺激を、実際にくふうして与えたいのです。中・高校生には少しレベルの高いハードルを用意することもたいせつで、そこから真剣な問い、生活の質への疑問も出てきます。たとえば「被服」の授業が食教育に、健康教育、マナーや伝統をたいせつにする教育にも連動して、生き生きした学びとなることを、生徒の成長から知らされました。

「ショーを終えた生徒の1人と礼拝堂の前で。まるで本当の結婚式に参加しているような気持ちになりました」

今月の先生

杉 信子

すぎのぶこ●横浜英和女学院中学高等学校家庭科、情報科教諭。給食室の管理栄養士と組んで食の家庭科教育にも力を入れている。

ベテラン家庭科教諭による
授業実践のお話

原 奈都子先生

家庭科教諭レポート③

「環境にやさしいくふう」の実体験

生きる力・活かす力……現代社会のニーズをふまえた家庭科とは？

読者の皆さん、家庭科は好きでしたか？

私の場合、中学校時代には家庭科は大の苦手。調理実習は大好きでしたが、被服実習に悩まされたことを思い出します。そんな私がなぜか34年間も家庭科教育に携わることとなりましたが、時代の流れとともに家庭科教育も大きく変化し、「裁縫・料理」というイメージから、男女共習で男女がともに学ぶ教科、「生きるために、たいせつな力を培う教科」として進化してきました。

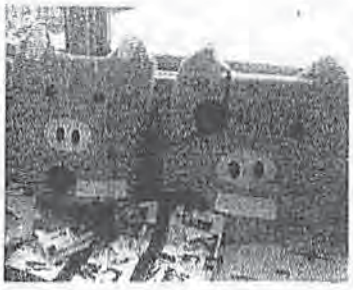
日ごろの授業では、衣・食・住の生活に密着した内容と、社会のニーズをあわせて学習できるように、またその内容を家庭生活に活かせるようにと考えています。

たとえば「環境にやさしいくふう」というテーマの調理実習では、「買い物」「調理」「後かたづけ」の過程を「環境・エコ・省エネ」を意識しながら行ないます。

「買い物」では、なるべく地域の小売店でトレーなど使い捨て容器や包装の少ないものを選び、地域の食材、たとえば地元産の小松菜を買うとか、エコバッグを持参してレジ袋をもらわないこと。「調理」ではゴミをできるだけ出さないよう、皮ごと食べられるにんじんなどの野菜は皮をむかないとか、三角コーナーをあえて置かないこと。「後かたづけ」では水を節約するために洗剤をうすめて使う、野菜のゆで汁を洗い物に活用する、よごれが落としやすい「アクリルたわし」を手作りするなど……。

〝ちょっとのくふう〟が環境を守ることを学んだ生徒たちは、「今日のゴミはこれだけ？」とゴミの少なさに驚き、「にんじんは皮も食べられるんだ！」と皮ごと作った料理のおいしさに感動します。実習では生徒同士のコミュニケーションをはかり、心を育てる家庭科でありたいと願っているので、そんな様子を見て、私も思わずうれしくなるものです。

生徒はもちろん、保護者の皆さんにも「家庭科って楽しい。ためになる」そう思っていただけることがなによりの喜びですが、将来、生徒たちが大人になったときに自立した生活が送れるよう「家庭科で習ったことが役立った」と感じられるような、そしていつも私自身がワクワクするような家庭科でありたいと、奮闘しています。



教室風景。実習で生徒たちが作った動物のざぶとんやトートバッグ。

今月の先生

原 奈都子

はらなつこ●江戸川区立小松川第二中学校家庭科教諭。「家庭科の中でなにか1つでも生徒の生きる力となり役に立てたらと思っています」

参　加　者　名　簿　（敬称略）

卒業生56名、在学生10名、現・旧教職員17名　合計83名

【卒業生】　（50音順）

荒井　麻由	里見　和子
阿彦　晴子	佐野　由佳
新井　佑佳	鹿田　裕美
飯塚　寛子	篠崎 佐枝子
石毛 妙子	柴崎 千佳子
石渡 光子	末武　舞
一見　恵子	杉 信子
伊藤 広子	瀬山　真美
井上 紗織	曾根田 美穂
今井　加奈子	高田　政子
岩崎 真知子	田島 夕貴絵
梅内　雅江	立川　まなみ
奥村 さえ子	立花　凡江
落合　充利	田村　京子
折茂　真美	中村　友紀
粕谷　和美	難波　宏子
叶内　茜	西村　美里
川野　美知子	丹羽 英子
岸　めぐみ	野尻 幸子
来栖　やよい	原 奈都子
桑原　福代	檜山 時恵
計良 智子	柵木　一美
小柳 陽子	又吉　美香
小山 素子	森　久美子
坂部 康子	森　実知子
櫻庭 洋子	八木　晶子
笹本 裕子	矢野 陽子
佐藤 美代子	渡邊 奈緒子

【在学生】

鈴木　久美子
青木　ななこ
小林　未佳
櫻下　美里
萩原　あずさ
本谷　麻笑
水野　徳子
森田　佑紀
久保田　幸子
中山　桃子

【旧教職員】

名誉教授	木村　廣子

【教職員】

女子栄養大学　学長	香川　芳子
女子栄養大学　副学長	香川　靖雄
女子栄養大学　副学長	五明　紀春
女子栄養大学　栄養学部長	安原　安代
香川栄養学園　常務理事	山根　正彦
教授	坂谷　幸恵
教授	井元　りえ
教授	島崎　とみ子
教授	仙波　圭子
教授	田中　みどり
准教授	平田　裕美
准教授	水崎　富美
実験実習助手	石川　梨都
実験実習助手	廣瀬　香織
大学教務学生部 大学就職担当	杉山　大
香友会	古川　知子

【プロジェクト・チーム】

井元りえ　板谷幸恵　仙波佳子

教職課程・家庭科委員会　石川梨都　廣瀬香織