bration: More new evidence from Spain. *Evolutionary Anthropology* 2:195–98.
STUTZ, A. 1993. Settlement patterns in Late Glacial northwestern Europe: The example from the Lesse Valley Magdalenian. B.A. honors thesis, Harvard University, Cambridge, Mass.
TEHEUX, E. 1994. Le Magdalénien de la Vallée de la Lesse. M.A. thesis, Université de Liège, Liège, Belgium.
TOUSSAINT, M. 1992. "The role of Wallonia in the history of palaeoanthropology," in *Cinq millions d'années: L'aventure humaine.* Edited by M. Toussaint, pp. 27–41. Liège: ERAUL.
VERMEERSCH, P., AND N. SYMENS. 1988. "Le Magdalénien de plein air en Belgique," in *De la Loire à l'Oder.* Edited by M. Otte, pp. 243–58. British Archaeological Reports S-444.

Childhood Stress and Family Environment[1]

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Human infants and juveniles cannot survive, let alone develop effective social skills, without assistance from parents or other caretakers. Relationships within the caretaking household are essential to the developing child (Bowlby 1969, 1973; Kagan 1984). The human mind is therefore likely to have evolved special sensitivity to interactions with family caretakers, particularly during infancy and early childhood (Daly and Wilson 1995).

Investigation of family environment and childhood stress has been hampered by the lack of noninvasive techniques for measurement of physiological stress response. Frequent collection of plasma or urine samples from children in nonclinical settings is not feasible. The development of saliva radioimmunoassay techniques (Hiramatsu 1981, Riad-Fahmy, Read, and Hughes 1983), however, presents new opportunities for stress research (e.g., Gunnar 1992, Kirschbaum and Hellhammer 1994). Saliva is relatively easy to collect and store, especially under the adverse field conditions faced by anthropologists (Ellison 1988). Concurrent monitoring of salivary cortisol levels and caretaker-child interactions provides a powerful research design for examining relations between childhood stress and family environment.

STRESS RESPONSE MECHANISMS AND THEORY

Stress response is modulated by the limbic system (amygdala and hippocampus) and basal ganglia which interact with two neuroendocrine axes: the sympathetic-adrenal medullary (SAM) system and the hypothalamic–anterior pituitary–adrenal cortex (HPA) system. The SAM and HPA systems affect a wide range of physiological functions in concert with other neuroendocrine mechanisms and involve complex feedback regulation. The SAM system controls the catecholamines norepinephrine and epinephrine (adrenalin). The HPA system regulates glucocorticoids, primarily cortisol (for reviews see Rose 1980, Gray 1987, Sapolsky 1992*a*).

Cortisol is a key hormone produced in response to psychosocial stress (Mason 1968, Selye 1976). Without cortisol, humans cannot endure the ups-and-downs of everyday life. Cortisol has a wide range of physiological effects, including (1) modulation of energy release (e.g., stimulation of hepatic gluconeogenesis in concert with glucagon and inhibition of the effects of insulin), (2) modulation of (and protection from?) immune activity, and (3) modulation of mental alertness, memorization, and learning (van Wimersma Greidanus and DeWied 1977, McGaugh 1989, Born et al. 1989, De Kloet 1991). Mechanisms for localized targeting (e.g., glucose uptake by active versus inactive muscle tissues and neuropeptide-directed immune response) provide fine-tuning of the above general physiological effects. Cortisol regulation allows the body to repond to changing environmental conditions by preparing for specific short-term demands perceived by the limbic system (Mason 1971, Munck, Guyre, and Holbrook 1984).

These temporary beneficial effects of stress response, however, are not without costs. Persistent activation of the HPA system is associated with immune deficiency, cognitive impairment, inhibited growth, delayed sexual maturity, damage to the hippocampus, and psychological maladjustment (Ader, Felton, and Cohen 1991; Dunn 1989, 1995; Glaser and Kiecolt-Glaser 1994). Deleterious consequences of chronic stress may involve compromised cellular energy metabolism (Atkinson 1977; Ivanovici and Wiebe 1981; Sapolsky 1986, 1991) and protection from autoimmunity (Munck, Guyre, and Holbrook 1984, Munck and Guyre 1991). Stressful life events—such as divorce, death of a family member, change of residence, or loss of a job—are associated with infectious disease and other health problems (House, Landis, and Umberson 1988, Kaplan 1991, Herbert and Cohen 1993, Maier, Watkins, and Fleshner 1994) (fig. 1).

Current psychosocial stress research suggests that cortisol response is stimulated by uncertainty that is perceived as significant[2] and for which behavioral responses

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2. In evolutionary terms, "significant" is used in the sense of a fitness-altering event. As a product of natural selection, cognitive

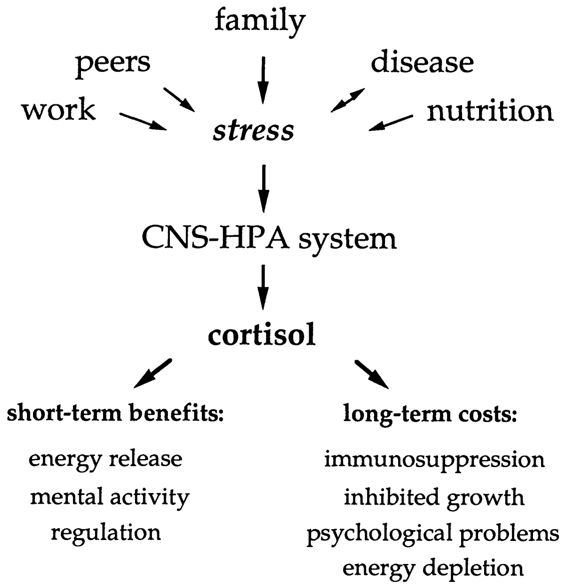

FIG. 1. *Human glucocorticoid stress response.*

will have unknown effects (Levine 1993, Kirschbaum and Hellhammer 1994, McEwen 1995). That is, important events are going to happen, and the child does not know how to react but is highly motivated to figure out what should be done. Cortisol release is associated with unpredictable, uncontrollable events that require full alert readiness and mental anticipation. In appropriate circumstances, temporary moderate increases in stress hormones (and associated neuropeptides) may enhance mental activity for short periods in localized areas, hence improving cognitive processes for responding to social challenges (cf. Coe et al. 1985, Martinez 1986, Breier et al. 1987, Martignoni et al. 1992). Mental processes unnecessary for appropriate response may be inhibited, perhaps to reduce external and internal "noise" (cf. Wolkowitz et al. 1990, Newcomer et al. 1994).

Relations between cortisol production and emotional distress are, however, difficult to assess because of temporal and interindividual variation in HPA response (Tennes and Mason 1982, Hubert and de Jong-Meyer 1989, Gunnar et al. 1992). Habituation may occur to repeated events for which a child acquires an effective mental model. Attenuation and below-normal levels of cortisol may follow a day or more after emotionally charged events. Chronically stressed children may develop abnormal cortisol response, possibly via changes in binding globulin levels and/or reduced affinity or density of glucocorticoid or CRH/vasopressin receptors in the brain (Fuchs and Flugge 1994). Early experience—such as perinatal stimulation of rats (Meaney et al. 1991), prenatal stress of rhesus macaques (Schneider, Coe, and Lubach 1992, Clarke 1993), prenatal exposure to cocaine among humans (Magnano, Gardner, and Karmel 1992), and sexual abuse among humans (De Bellis et al. 1994)—may permanently alter HPA response. And personality may affect HPA response (and vice versa) because children with inhibited temperaments tend to have higher cortisol levels than extraverted children (Kagan, Resnick, and Snidman 1988, cf. Flinn, England, and Beer 1992).

To further complicate matters, HPA stress response involves a wide variety of other neuroendocrine activities, including modulation of catecholamines, corticotropin-releasing hormone (CRH), melatonin, testosterone, serotonin, β-endorphins, cytokines, and enkephalins (Axelrod and Reisine 1984, Asterita 1985, De Kloet 1991, Sapolsky 1992*a*, Saphier et al. 1994). Changes in cortisol for energy allocation and modulation of immune function may be confused with effects of psychosocial stress. Intracerebral oxytocin and vasopressin are associated with familial attachment (Insel and Shapiro 1992, Levy et al. 1992, Winslow et al. 1993) and may influence distress involving caretaker-child relationships. Concurrent monitoring of all of these neuroendocrine activities would provide important information about stress response but is impossible in a nonclinical setting with current techniques.

In sum, assessment of relations between naturally occurring emotional states and cortisol is complex, requiring longitudinal monitoring of hormone levels, control of extraneous effects from physical activity, circadian rhythms, and food consumption, knowledge of individual differences in temperament, experience, and perception, and awareness of cultural context. Anthropological research that integrates biology and ethnography is particularly well suited to these demands (e.g., Armelagos et al. 1992; Brown 1981, 1982; Ellison 1994; Goodman et al. 1988; Hanna, James, and Martz 1986; James, Crews, and Pearson 1989; Pearson, James, and Brown 1993; Weisner and Abbott 1977; Weisner 1981; Werner 1985). Physiological assessment in concert with ethnography and coresidence with children and their families in a small village over an eight-year period can provide intimate, prospective, naturalistic information unavailable to clinical studies.

RESEARCH DESIGN

Longitudinal monitoring of cortisol in a natural (nonclinical) environment is used to identify specific psychosocial causes and consequences of childhood stress. Data analyses examine both long-term (cumulative) and short-term (day-to-day, hour-by-hour) associations among cortisol levels, family composition, behavioral activities, events, temperament, immune response, and health.

Variables and measures are as follows: *Physiological stress response* is assessed by radioimmunoassay of cortisol levels in saliva. Analyses examine mean values, variation, and day-to-day and hour-by-hour profiles of standardized (circadian time control) cortisol data. *Fam-*

processing of information in regard to "stress" involves assessment of fitness consequences of potential "stressors." "Positive" or "negative" affect is irrelevant. The criterion is whether stimulation of stress response would be advantageous (fitness-maximizing).

ily environment is assessed by (*a*) age, sex, genealogical relationship, and number of individuals in the household (family composition) and (*b*) household income, material possessions, landownership, occupations, and educational attainment. *Caretaking attention* is assessed by (*a*) frequencies and types of behavioral interaction, (*b*) informant ratings of caretaking that children receive, and (*c*) informant interviews. *Personality and temperament* are assessed by (*a*) culturally appropriate versions of EAS (Buss and Plomin 1984), five-factor (Goldberg 1992, 1993), and DOTS-R (Windle and Lerner 1986) instruments, (*b*) informant (peers, parents, teachers, neighbors) interviews, and (*c*) behavioral observation. *Immune response* is assessed by radioimmunoassay of interleukin-8 and neopterin, turbidimetric immunoassay of secretory immunoglobulin A, and microparticle enzyme immunoassay of microglobulin β2 from saliva samples. *Health* is assessed by (*a*) observed type, frequency, and severity of medical problems (diarrhea, influenza, common cold, asthma, abrasions, rashes, etc.), (*b*) informant (parents, teachers, neighbors) ratings, (*c*) medical records, and (*d*) physical examination by a medical doctor. *Daily activities* and *emotional states* are assessed from (*a*) caretaker and child self-report questionnaires and (*b*) systematic behavioral observation (focal follow and instantaneous scan sampling). Multiple sources of information are cross-checked to assess reliability (Bernard et al. 1984).

The primary focus of this report is on relations between stress (dependent variable) and family environment (independent variable). Caretaking attention, temperament, immune function, health, daily activities, and emotional states are analyzed as secondary or control variables and are presented in more detail in other publications.

STUDY SITE

"Bwa Mawego"[3] is a rural village located on the east coast of Dominica. The 780± residents live in 206± structures/households that are loosely clumped into five "hamlets" or neighborhoods. The population is of mixed African, Carib, and European descent. The village is isolated because it sits at the dead-end of a rough road passable by small trucks except for occasional periods during the rainy season (the road was improved and partially paved in 1991 and 1993). Part-time residence is common, with many individuals emigrating for temporary work to other parts of Dominica or off-island (e.g., seasonal farm work in the United States and Canada). Most residents cultivate bananas and/or bay leaves as cash crops and plantains, dasheen, and a variety of fruits and vegetables as subsistence crops. Fish are caught by free-diving with spear-guns and from small boats (hand-built wooden "canoes" of Carib design) using lines and nets. Land is communally "owned" by kin groups but parcelled out for long-term individual use.

Most village houses are strung close together along roads and tracks. Older homes are constructed of wooden planks and shingles hewn by hand from local forest trees; concrete block and galvanized roofing are more popular today. Most houses have one or two sleeping rooms, with the kitchen and toilet as outbuildings. Children usually sleep together on foam or rag mats. Wealthier households typically have "parlors" with sitting furniture. Electricity became available in 1988; today about 60% of homes have "current," 23% have telephones, 11% have refrigerators, and 7% have televisions. Water is obtained from streams, spring catchments, and run-off from roofs.

The study involves 247 children aged 2 months–18 years residing in 82 households. This is a nearly complete sample (>95%) of the children living in four of the five village hamlets during the period of fieldwork. Research was conducted during June–August 1988, June–August 1989, May–December 1990, May–August 1992, June–August 1993, December 1993–February 1994, May–August 1994, and June–August 1995.[4] Cortisol data are from saliva samples collected during July–December 1990, June–August 1992, June–August 1993, December 1993–January 1994, and June–August 1994.[5]

METHODS AND FIELD TECHNIQUES

Information on household environment, caretaking attention, temperament, and health was collected by standard ethnographic techniques including interviews, behavioral scans, participant observation, and questionnaire instruments. These methods are described in more detail in other publications (Flinn 1988, Quinlan n.d.)

Data on physiological stress response are derived from radioimmunoassay of saliva samples. Saliva was collected by two routines. The primary routine was a twice-daily collection in which an anthropologist and research assistant walked set routes from house to house, once in the morning (5:30 A.M.–9:00 A.M.) and once in the afternoon (3:00 P.M.–6:30 P.M.). Most (16,652 of 18,376) saliva samples were collected this way. The second routine used a "focal follow" technique in which (*a*) the

3. Pseudonyms are used for confidentiality.

4. MVF was assisted in the field by Eric Durbrow in 1988, 1989, and 1990, Ingrid Bozoky, Tomasz Beer, and Carol Ward in 1990, Seamus Decker in 1992, 1993, and 1994, David Tedeschi in 1992 and 1993, Robert Quinlan and Marsha Quinlan in 1993 and 1994, and Mark Turner in 1993, 1994, and 1995.

5. During August–December 1990 saliva was collected twice daily from each child for four six-day periods. Because saliva must be collected during a limited time period, the study population was divided into two roughly equal halves, with collection periods alternating between them (i.e., six days with one half, then six days with the other); hence saliva was collected for a total of four + four = eight six-day periods. Day-long focal follows with hourly saliva collection were conducted with two children to provide more precise information on cortisol variation during the day. During the 1992 field season, MVF was assisted by two graduate students, and saliva was collected concurrently from all children during two six-day periods. During the 1993 and 1994 field seasons there were three graduate student assistants, and saliva was collected concurrently from all children during six six-day periods. Day-long focal follows were conducted with four children. Additional morning-long focal follows were conducted with 22 infants and mothers.

child was observed from dawn until early afternoon or evening or (*b*) the infant was observed from dawn until early afternoon, both with hourly saliva samples (1,724 of 18,376).

Our saliva collection protocol was as follows: First, each child rinsed her/his mouth with fresh water. At this point, children were checked for oral bleeding. Both food and blood contamination may affect the integrity of samples (Ellison 1988). Next, children were given 1/4–1/2 stick of Wrigley's gum (spearmint) to stimulate saliva production. After chewing the gum for about one minute and "swallowing the sugar," saliva was deposited in disposable plastic cups for about three minutes. For infants, saliva was collected by swabbing with cotton rolls (Turner n.d.). Approximately 4 ml of saliva was pipetted into labeled (name, date, time, number) polystyrene test tubes and preserved using sodium azide and refrigeration. Analysis of cortisol levels requires precise information on time of collection, time of waking up from sleep, and individual sleep schedule because there is a circadian pattern to cortisol release (Van Cauter 1990). Some hourly samples were taken for finer-grained analysis of temporal fluctuation in cortisol levels. Daily activity, emotional state, and health questionnaires were administered concomitant with saliva collection.

Children readily adjusted to the collection procedure. However, shy or inhibited children in particular tended to have cortisol levels higher than normal for the first few days of saliva collection. Multiple samples (more than 50 days of morning and afternoon samples) from each child provided a more effective indication of stress response than a single-collection design.

Cortisol levels in saliva samples were determined by radioimmunoassay. Salivary cortisol is a reliable measure of adrenal corticol function (Hiramatsu 1981, Umeda et al. 1981, Vining et al. 1983, Landon et al. 1984, Bolufer et al. 1989). All samples were analyzed at the Ligand Assay Laboratory at the University of Michigan Hospitals.

Our radioimmunoassay protocol was as follows: (1) Saliva tubes were decapped and centrifuged at 2,400 rpm for 10 minutes at 6° C. (2) The tubes were placed in a rack for an automatic pipetting robot, which pipetted 200 μl of saliva from each sample tube into receiver tubes from DPC I^{125} cortisol solid-phase radioimmunoassay kits. Each sample was duplicated. (3) The radioactive label was added, and the tubes were vortexed. (4) The receiver tubes were placed in 37° C water baths for 45 minutes, after which they were aspirated (twice). (5) The receiver tubes were run through a gamma counter. (6) Data from the counter were analyzed with a statistical program (StatLIA). Six standards (.25 μg/dl–50 μg/dl) were used to determine the assay curve. B_0/B_{100} ratios, covariance of duplicates, and interassay variation using these techniques were of high quality (e.g., covariance of duplicates for 1993 samples averaged 1.8%).

Cortisol release follows a circadian pattern, with highest levels around waking up in the morning, diminishing to low levels during the evening hours and a nadir during sleep. For example, mean cortisol level when children have been awake for ten minutes is 4.63 μg/dl; for two hours it is 1.78 μg/dl, and for eight hours it is .57 μg/dl. To control for these circadian effects, raw cortisol measures were standardized by five-minute time intervals.[6] For each time interval, mean values and standard deviations were computed. Standardized values were generated at time-controlled measures of cortisol response as with the following hypothetical example: the mean cortisol level of children who have been awake for 60 minutes is 3.0 μg/dl, with a standard deviation of 1.0 μg/dl. A cortisol measure of 4.0 μg/dl from this time period would have a standardized value of 1.0 (i.e., 4.0 μg/dl is one standard deviation [1.0 μg/dl] from the average [mean] value of 3.0 μg/dl). This procedure allows comparison of cortisol values from saliva collected at different times. All cortisol data presented in this paper are time-standardized.

During saliva collection, children and their caretakers were asked a series of questions concerning the child's activities that day and how the child felt during these activities. This self-report and caretaker-report information on daily activities was compared with behavioral observation data. Health evaluations were also conducted daily during saliva collection. If illness was indicated, body temperature was checked using an oral thermometer. Blood pressure was measured once a week.[7]

Most individuals exhibit a slight rise (about .1 μg/dl at lunch, or 18%) in cortisol levels after eating a meal or drinking caffeinated beverages. This postprandial rise is most significant for the midday meal. Data were not adjusted for eating and caffeine intake because very few samples were taken during lunchtime, the effect is small, and the occurrence of eating and caffeine intake was presumed random with regard to the hypotheses tested. Individuals commonly have small elevations in cortisol levels from midmorning to midafternoon, usually in association with potential minor stressors (Holl et al. 1984).

Some individuals show a slight rise in cortisol levels during and shortly after intensive physical exertion (e.g., carrying heavy loads of wood, water, bananas, or bay leaf). Physical exertion involving social interaction, such as competitive sports, is associated with more substantial elevation of cortisol levels, particularly for males. Data were not adjusted for physical activity because only a small proportion of samples were collected during physical exertion, it was difficult to determine the degree of this exertion, and such activities were presumed random with regard to the hypotheses tested. Because general activity levels are associated with cortisol,

6. Wake-up time was used because individuals had different sleep schedules; some children habitually arose at 5:30 A.M., whereas others slept until 7:00 A.M. or later. Five-minute time intervals were necessary because of the quick drop in cortisol levels during the morning (approximately a one-hour half-life, from about 4.5 μg/dl at 30 minutes after wake-up to 2 μg/dl at 120 minutes after wake-up). This technique required a large number of samples (>100 samples per time interval).

7. Blood pressure was measured (consistently) during the 1990 and 1992 field seasons only.

TABLE 1
Household Composition (Primary Caretakers) of Study Sample

Household Composition	Number (and Percentage) of Children in Sample		
	1990–91	1992	1993–94
Bi-parental ("nuclear")	69 (33.0)	81 (34.9)	86 (36.1)
Bi-parental, father absent >50%	24 (11.5)	14 (6.0)	16 (6.7)
Single mother	19 (9.1)	27 (11.6)	27 (11.3)
Single mother + kin	24 (11.5)	28 (12.1)	25 (10.5)
Single father	3 (1.4)	2 (0.9)	1 (0.4)
Grandmother	26 (12.4)	28 (12.1)	29 (12.2)
Grandparents	14 (6.7)	16 (6.9)	16 (6.7)
Other kin (aunt, cousin)	4 (1.9)	6 (2.6)	6 (2.5)
Mother + stepfather w/o children	3 (1.4)	3 (1.3)	3 (1.3)
Mother + stepfather w/children	19 (9.1)	22 (9.5)	24 (10.1)
Nonrelative	7 (3.3)	5 (2.2)	5 (2.1)

this presents a confounding effect. Some children may have increased cortisol response when they are healthy, active, and have abundant energetic resources compared with when they are inactive and have depleted energy reserves.

RESULTS: CHILDHOOD STRESS AND HOUSEHOLD COMPOSITION

Children in Bwa Mawego live in a variety of family environments. Table 1 provides information on the frequencies of 13 general types of household composition of the study sample during the field seasons of 1990–91, 1992, and 1993–94. Household composition in the Caribbean is dynamic; in Bwa Mawego during 1990–94, 31% of households with children changed composition (category) at least once, and many children (27%) resided in more than one household. "Household" is defined as a physical structure (house) in which a family resides (sleeps, eat meals, store possessions, etc.). The social and physical reality of caretaking households varies. Most have extensive kin ties to other households, often resulting in a "compound" of neighboring families. Others are more isolated. Additional important aspects of a child's family environment may include parents' marital relations, ties to relatives residing in other households, and use of alcohol by one or more caretakers.

Associations between average cortisol levels of children and household composition are presented in figure 2. These data indicate that children living with nonrelatives, stepfathers and half-siblings (stepfather has children by the stepchild's mother), or single parents without kin support have significantly higher average levels of cortisol than children living with both parents, single

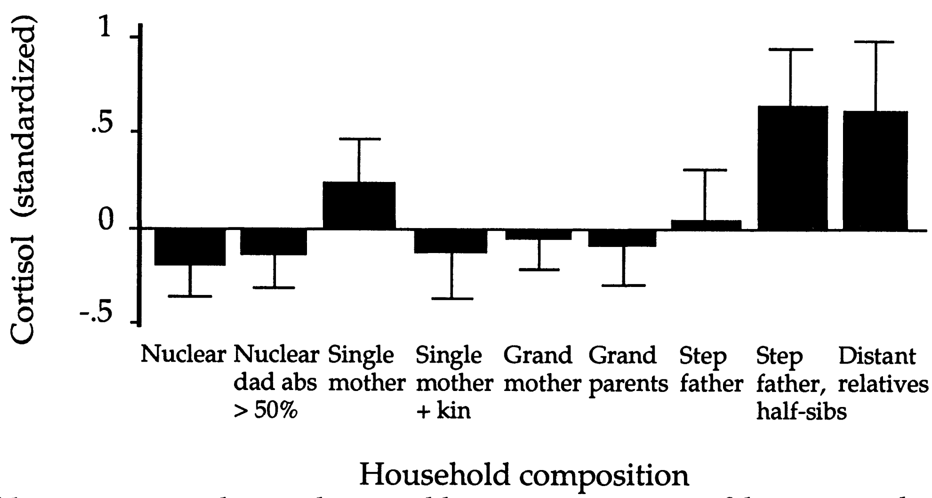

FIG. 2. *Household composition and cortisol. Vertical bars represent 95% confidence intervals (1.96 S.E.). Sample sizes (number of children, number of cortisol saliva assays) are 88, 6,397; 26, 1,930; 29, 2,078; 31, 2,327; 32, 2,429; 16, 1,235; 5, 244; 24, 1,648; 9, 434.*

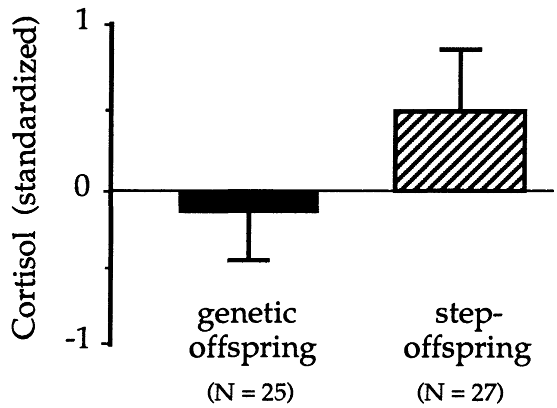

FIG. 3. *Average (mean) cortisol levels of step- and genetic children residing in the same household. Average age of stepchildren is 10.3 years, that of genetic children 6.4 years. Vertical lines indicate 99% confidence intervals (p < .01).*

mothers with kin support, or grandparents.[8] A further test of this hypothesis is provided by comparison of step- and genetic children residing in the same households (fig. 3). These data indicate that stepchildren have higher average cortisol levels than their half-siblings residing in the same household who are genetic offspring of both parents.

We conclude from these data that childhood stress is associated with household composition. There are several possible reasons. Children in difficult caretaking environments may experience chronic stress resulting in moderate-to-high levels of cortisol (i.e., a child has cortisol levels that are above average day after day). They may experience more acute stressors that substantially raise cortisol for short periods of time. They may experience more frequent stressful events (e.g., parental chastisement or marital quarreling [see Wilson, Daly, and Weghorst 1980, Flinn 1988, Finkelhor and Dzuiba-Leatherman 1994) that temporarily raise cortisol. There may be a lack of parental consolation or reconciliation. And they may have inadequate coping abilities, perhaps resulting from difficult experiences in early development. The following case examples present temporal analyses of family relations and cortisol levels that illustrate some of the above possibilities.

"Polly" is a nine-year-old girl who lives with her grandmother, three aunts, and 5–12 cousins. Her mother has been away working in a hotel on St. Maarten for the past two years. At 4:17 P.M. on September 17, 1992, MVF observed the following events: Polly spilled a small cup of water inside the house. Her aunt responded by yelling at Polly and swinging a hand at her back. Polly ducked away and hid under the kitchen table. Her aunt continued to scold her and threatened that she (Polly) would have to leave and live somewhere else if she did not become less troublesome. Polly hid her face and began to cry. She stayed under the table for the next ten minutes until her aunt left the house.

Polly's cortisol levels were elevated for the next two days, followed by subnormal levels (a possible recovery period?). Four days later she reported feeling ill and had a runny nose and oral temperature of 100.3 (fig. 4). In 1993 she moved from her grandmother's home to her great-grandparents'. Her cortisol levels have been more moderate (mean = 0.8, down from 1.43) and less variable (standard deviation = .27, down from .44) since this move. Now Polly more frequently reports feeling happy, is more frequently observed smiling, is less frequently observed withdrawing or hiding her face, and has less frequent illness.

On September 28, 1990, a serious marital quarrel erupted in the "Belles" household. "Aretha" is a 37-year-old mother of five children, three of whom (ages 10, 12, and 14) are living with her and their father, "Arnie Belles." Aretha was angry with Arnie for reportedly seeing another woman and left the village to stay with her sister in Martinique for 13 months. She returned in 1991 and reconciled with Arnie, with whom she is currently living happily. Their coresident children showed elevated cortisol levels for a prolonged period following their mother's departure (fig. 5). Children usually habituate to most stressful events, but absence of a mother often results in an abnormal pattern of long-term elevated cortisol levels. Following the return of their mother, the children's cortisol levels resumed a more normal profile. Children living in families with high levels of marital conflict (observed and reported serious

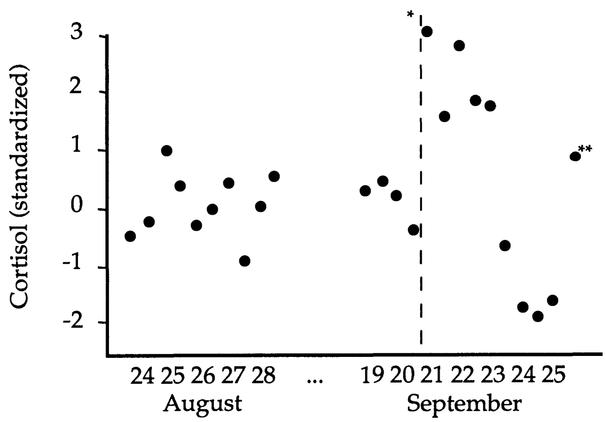

FIG. 4. *Morning and afternoon cortisol levels for Polly during 1992. Late August cortisol levels are normal, but after being threatened by her aunt on the afternoon of September 20 (*) she has elevated cortisol levels for two days, followed by depressed cortisol levels for two days. She exhibits symptoms of a common cold on the afternoon of September 25 (**).*

8. The relation between cortisol levels and household composition for infants (one to fourteen months) is less certain. Preliminary analyses indicate that infants in households without fathers have lower cortisol levels than infants in households with coresident fathers (Turner, Flinn, and Edwards 1995). However, this may reflect higher activity levels, different sleeping patterns, and/or more frequent breast feeding among father-resident infants rather than higher levels of psycho-social stress.

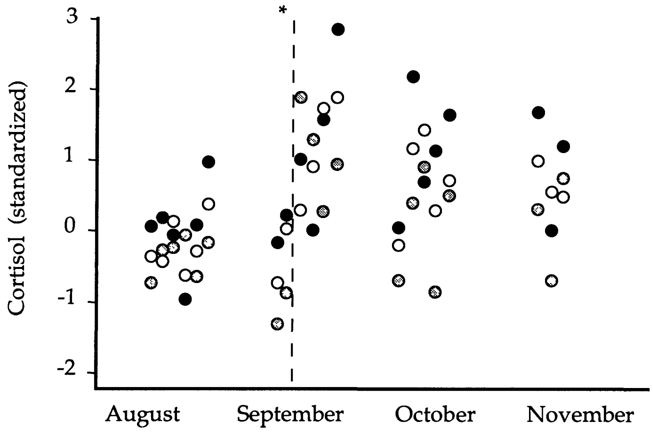

FIG. 5. *Marital conflict and cortisol levels in the Belles family.* ●, *10-year-old female;* ◎, *12-year-old male;* ○, *14-year-old male. Mean cortisol level before parental quarrel* (*) *was* −.37*; for two months thereafter it was* 1.42 ($p < .01$).

quarreling, fighting, residence absence) usually have higher average cortisol levels than children living in more amiable families.

The events in the children's lives that are associated with elevated cortisol are not always traumatic or even "negative." Eating meals, hard physical work, routine competitive play such as cricket, basketball, and "king of the mountain" on ocean rocks, and return of a family member who was temporarily absent (e.g., father returning from a job in town for the weekend) are associated with temporary moderate increases (about 10%–100%) in cortisol among healthy children. These moderate stressors—"arousers" might be a more appropriate term—usually have rapid attenuation (< one hour) of cortisol levels. Some stressors/arousers have characteristic "signatures" of cortisol level and duration.

High-stress events (cortisol increases from 100% to 2,000%), however, most commonly involve trauma from family conflict or change. Punishment, quarreling, and residence change substantially increase cortisol levels, whereas calm affectionate contact is associated with diminished (−10% to −50%) cortisol levels (table 2). Of the cortisol values that are more than two standard deviations above mean levels (i.e., indicative of substantial stress), 19.2% are temporally associated with traumatic family events (residence change of child or parent/caretaker, punishment, "shame," serious quarreling, and/or fighting); 47.4% of traumatic family events are temporally associated with substantially elevated cortisol (> two standard deviations above mean levels). Although there are age differences in stress response to family conflict (Flinn et al. 1995*b*), traumatic family events are associated with elevated cortisol levels for all ages of children more than any other factor that we examined. These results indicate that family interactions are a critical psychosocial stressor in children's lives.

Although elevated cortisol levels are associated with traumatic events such as family conflict, long-term stress may result in diminished cortisol response. In some cases chronically stressed children have blunted response to physical activities that normally evoke cortisol elevation. Comparison of cortisol levels during "nonstressful" periods (no reported or observed crying, punishment, anxiety, residence change, family conflict, or health problem during 24-hour period before saliva collection) indicates a striking reduction and, in many cases, reversal of the family environment–cortisol association (fig. 6). Chronically stressed children commonly have subnormal cortisol levels when they are not in stressful situations. For example, cortisol levels immediately after school (walking home from school) and during noncompetitive play are lower among some chronically stressed children (cf. Long, Ungpakorn, and Harrison 1993).

Abnormal cortisol response may involve immunosuppression. Some chronically stressed children appear to have diminished cell-mediated (neopterin, microglobulin β2), humoral (secretory-immunoglobulin A), and/or nonspecific (neutrophil recruitment via interleukin-8) immunity (Flinn et al. 1995*a*, in preparation). Stress response may deplete cellular energy and immune reserves (perhaps including protection from autoimmunity) that require subsequent conservation to rebuild normal levels. Although cortisol may provide short-term energy release benefits, the body needs to replenish energy reserves to provide for immunity, growth, and other functions. Chronic stress and high average cortisol levels are associated with frequency of illness (fig. 7), a stress–health relation suggested by temporal associations such as the above example of Polly, in which illness followed several days after a high-stress event (see also Mason et al. 1979; Coe, Lubach, and Ershler 1989; Cohen, Tyrrell, and Smith 1991, 1993; Stone et al. 1992; Evans and Pitt 1994). This association between illness and cortisol is significant within as well as among households.

Longitudinal analysis of caretaking histories indicates that children may have "sensitive periods" for development of stress response. Children with severe caretaking problems during infancy (neglect, parental alcoholism, and/or maternal absence) frequently exhibit one of two distinct cortisol profiles: (1) unusually low basal cortisol levels with occasional high spikes (fig. 8) or (2) chronically high cortisol levels (fig. 9). The first type (low basal with high spikes) is associated with antisocial and delinquency behavior (e.g., theft, running away from home) and is more common among males. The second type (chronically high) is associated with anxiety and withdrawal behavior and is more common among females. These profiles are suggestive of diminished glucocorticoid regulatory function of the hippocampus (cf. Gray 1987, Sapolsky 1992*a*, Yehuda et al. 1991).

"George" is a 13-year-old boy who lives most of the year with his father. He recently stopped attending school and sometimes does not come home at night, and he has been accused of stealing money from an elderly

TABLE 2
Levels of Cortisol for Events and Activities during Eight-Hour Period Preceding Collection of Saliva

Event/Activity	Frequency (%)	Mean Cortisol (Standardized)	p
"Traumatic" family events			
Severe punishment	0.4	1.72	.0001
Family fight	2.3	1.09	.001
Residence change	2.7	0.68	.01
Moderate punishment	1.1	0.60	.01
Family alcohol	3.2	0.58	.05
"Shamed"	1.6	0.55	.01
"Normal" family activities			
Mild punishment	6.4	0.14	n.s
Domestic chores	22.6	−0.06	n.s.
Sibling child care	9.8	−0.11	n.s.
"Affectionate" contact	2.4	−0.26	n.s.
Caretaker trip to town	4.7	0.08	n.s.
Health conditions			
Common cold, fever	0.6	0.96	.001
Common cold, no fever	2.2	0.27	n.s.
Diarrhea, gas, worms	0.5	0.10	n.s.
Moderate injury	0.4	0.62	.05
Lethargic	0.8	0.03	n.s.
Physical and social activities			
Hard work (current)	0.5	0.52	.05
Hard work (>1 hr. ago)	3.1	0.12	n.s.
Moderate work (current)	3.8	0.04	n.s.
Moderate work (>1 hr. ago)	17.3	−0.06	n.s.
Competitive play (current)	4.3	0.77	.05
Competitive play (>1 hr. ago)	7.6	0.08	n.s.
General play	28.2	−0.09	n.s.
School, normal	7.6	−0.04	n.s.
School, minor test	2.2	−0.05	n.s.
School, major test	0.3	0.41	n.s.
Fight w/peer (major)	0.2	0.65	.05
Fight w/peer (minor)	0.9	0.08	n.s.
Quarrel w/peer (major)	0.4	0.72	.01
Quarrel w/peer (minor)	1.0	0.21	n.s.

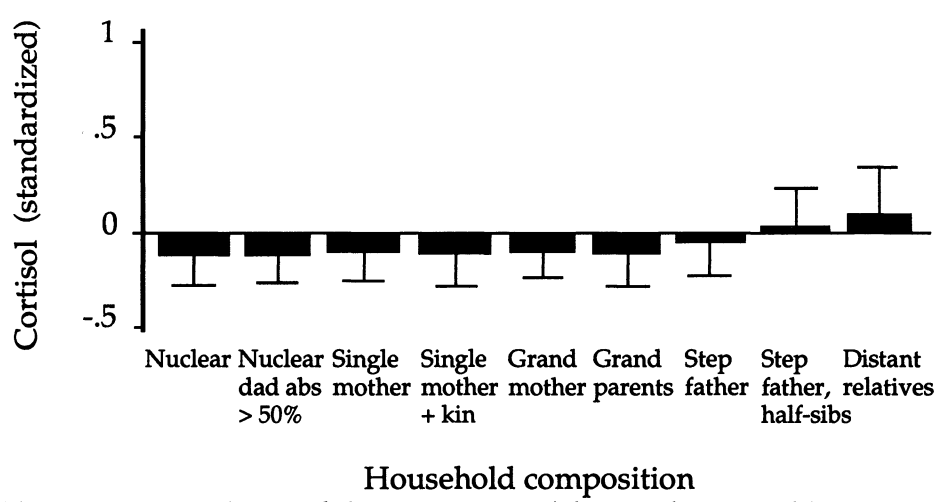

FIG. 6. *Household composition and cortisol during "nonstressful" periods. Vertical bars represent 95% confidence intervals (1.96 S.E.). Sample sizes (number of children, number of cortisol saliva assays) are 88, 4,122; 26, 1,116; 29, 1,140; 31, 1,479; 32, 1,638; 16, 884; 5, 163; 24, 792; 9, 244). There are no significant differences among household types.*

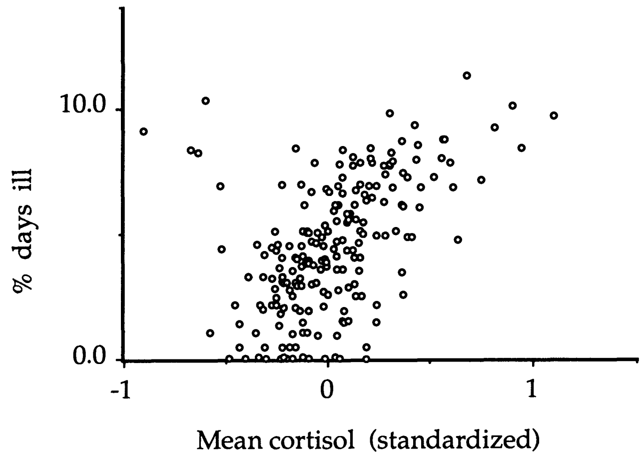

FIG. 7. *Average cortisol levels and frequency of illness during 11 months of direct observation. Samples collected when child was ill are excluded from computation of average cortisol values because cortisol levels tends to be higher during illness (Flinn et al. 1995a).* $R^2 = .522$, $p < .001$, $y = -.386 + .039x$.

woman. George had a difficult childhood and spent little time living with his mother. He does not have close friends and is usually alone or on the periphery of play groups. His cortisol levels are usually below normal but occasionally are very high. Four of 13 children (all 4 of them boys) with caretaking problems during infancy exhibit this low-basal-with-high-spikes cortisol profile. All exhibit problems that are typical of children with high cortisol profiles, including frequent illness, fatigue, reduced play activity, and sleep disturbances. It is possible that their low cortisol levels involve "recovery" from damage caused by chronic or acute stress, although the long-term persistence of this profile among some children suggests a more permanent organic basis, perhaps developed during infancy and/or in utero (cf. Cohen et al. 1989, Insel 1990).

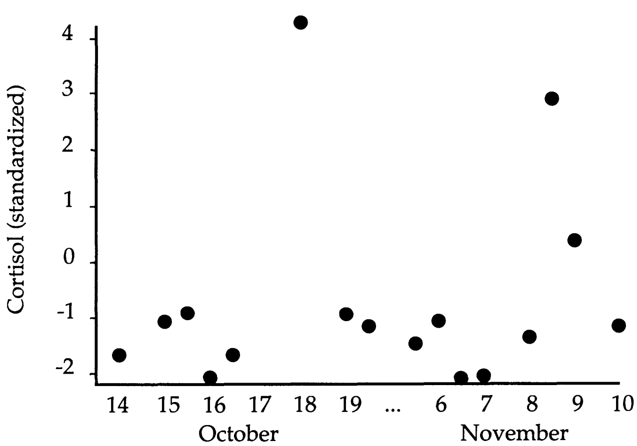

FIG. 8. *Cortisol profile of George, characterized by below-normal basal levels with occasional high spikes.*

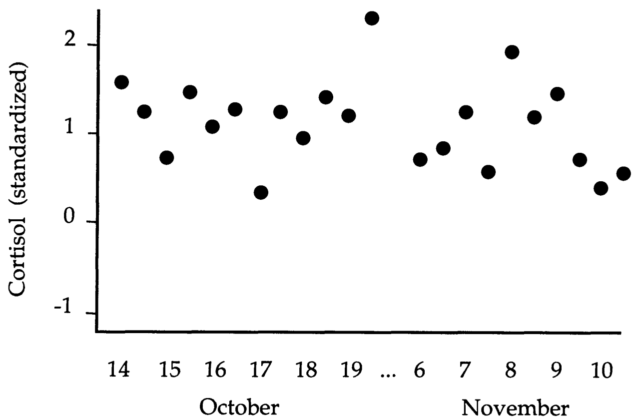

FIG. 9. *Cortisol profile of Louise, characterized by consistently high levels.*

"Louise" is a ten-year-old girl who lives with her mother, half-siblings, and stepfather. She is slim, shy, and quiet in comparison with most children, including her half-siblings. Her mother had a difficult time caring for Louise when she was an infant. Louise's father refused to acknowledge her as his child, and it was hard for Louise's mother to support her while establishing a new relationship with her current spouse. Louise had several growth disruptions during infancy and is ill more frequently than most children. Her cortisol levels are consistently above normal, with few apparent "recovery periods." Particularly high levels are usually associated with events that involve social anxiety (e.g., first day of school). Six of 13 children (5 girls and 1 boy) with caretaking problems during infancy exhibit this high cortisol profile.

These unusual cortisol profiles associated with caretaking problems during infancy may interact with personality development. All of the children with the low-basal-with-high-spikes cortisol profile have low sociability and high aggressiveness, whereas shyness and social anxiety is characteristic of children with the chronically high cortisol profile (cf. McBurnett et al. 1991).[9] Stepchildren of both sexes are also more likely to have unusual cortisol profiles and inhibited temperaments than their half-siblings residing in the same household who are genetic offspring of both parents (Flinn, England, and Beer 1992, Flinn et al. 1995a). Some children exhibit longitudinal change in cortisol profiles and personality concomitant with an improved family environment, but others do not. In contrast to their responses to most potential stressors, children do not seem to habituate readily to family trauma. Some parental actions may lack the predictability and controllability necessary for the development of actions or perceptions that reliably alleviate stress response (cf. Garmezy 1983, Gottman and Katz 1989).

9. These behavioral and personality characteristics probably involve other neuroendocrine systems, such as serotonin modulation (Brown et al. 1982).

SUMMARY AND CONCLUDING REMARKS

Stress response may be viewed as an adaptive mechanism that allocates energy resources to different bodily functions, including immunity, growth, muscle action, and cognition (Maier, Watkins, and Fleshner 1994, Sapolsky 1994, McEwen 1995). Understanding the algorithms by which stress response makes allocation decisions is important because of the consequences for health and psychological development (Tinbergen 1974).

The family is the most critical source of physical and social resources for children. Throughout human evolutionary history, parents and close relatives provided calories, protection, and information necessary for survival, growth, health, social success, and eventual reproduction (Alexander 1974, Konner 1981, Lancaster and Lancaster 1987). Natural selection is therefore likely to have favored particular sensitivity to interactions with family caretakers (Petrovich and Gewirtz 1985; Daly and Wilson 1988, 1995). Release of cortisol and other stress hormones in response to traumatic family events may modulate energy and mental activity to resolve perceived psychosocial problems.

The objective of our long-term ethnographic study in Bwa Mawego is to monitor children's social and physical environment, behavioral activities, health, mental perceptions, and physiological states in a naturalistic setting the better to understand relations between family environment and stress response. Analyses of data indicate that children living in households with intensive, stable caretaking usually have moderate cortisol levels. Children living in households with nonintensive, unstable caretaking are more likely to have abnormal (usually high and variable but sometimes low) cortisol levels. Traumatic family events are associated with elevated cortisol levels. Most children with caretaking problems during infancy have unusual cortisol profiles. These associations indicate that family environment is a significant source of stress for children living in Bwa Mawego. The variability of stress response, however, suggests a complex mix of each child's perceptions, neuroendocrinology, temperament, and specific context (see also Montager et al. 1978*a, b;* Montager, Restoin, and Henry 1982).

Relations between family environment and cortisol stress response appear to result from a combination of factors, including frequency of traumatic events, frequency of positive "affectionate" interactions, frequency of negative interactions such as irrational punishment, frequency of residence change, security of "attachment," development of coping abilities, and availability or intensity of caretaking attention. Probably the most important correlate of household composition that affects childhood stress is maternal care. Mothers in socially "secure" households (i.e., permanent coresidence with mate and/or other kin) appear more able and more motivated to provide child care. Mothers without mate or kin support are likely to exert effort attracting potential mates and may view dependent children as impediments to this. Hence coresidence of father may not only provide direct benefits from paternal care but also affect maternal care (Lamb et al. 1987, Scheper-Hughes 1988, Belsky, Steinberg, and Draper 1991, Flinn 1992, Hewlett 1992, Hurtado and Hill 1992). Young mothers without mate support usually rely extensively upon their parents or other kin for help with child care. Children born and raised in household environments in which mothers have little or no mate and kin support are at greatest risk for abnormal cortisol profiles and associated health problems. In comparison with Western industrialized societies, rural Caribbean communities are fortunate to have extensive kinship networks that provide alternative sources of child care and support for mothers despite difficult economic circumstances.

References Cited

ADER, R., D. L. FELTEN, AND N. COHEN. Editors. 1991. *Psychoneuroimmunology.* San Diego: Academic Press.

ALEXANDER, R. D. 1974. The evolution of social behavior. *Annual Review of Ecology and Systematics* 5:325–83.

ARMELAGOS, G. J., T. LEATHERMAN, M. RYAN, AND L. SIBLEY. 1992. Biocultural synthesis in medical anthropology. *Medical Anthropology* 14:35–52.

ASTERITA, M. E. 1985. *The physiology of stress.* New York: Human Services Press.

ATKINSON, D. E. 1977. *Cellular energy metabolism and its regulation.* New York: Academic Press.

AXELROD, J., AND T. D. REISINE. 1984. Stress hormones: Their interaction and regulation. *Science* 224:452–59.

BELSKY, J., L. STEINBERG, AND P. DRAPER. 1991. Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. *Child Development* 62:647–70.

BERNARD, H. R., P. D. KILLWORTH, D. KRONENFELD, AND L. SAILER. 1984. The problem of informant accuracy: The validity of retrospective data. *Annual Review of Anthropology* 13:495–517.

BOLUFER, P., A. GANDIA, A. RODRIGUEZ, AND P. ANTONIO. 1989. Salivary corticosteroids in the study of adrenal function. *Clinica Chimica Acta* 183:217–26.

BORN, J., V. HITZLER, R. PIETROWSKY, P. PAUSCHINGER, AND H. L. FEHM. 1989. Influences of cortisol on auditory evoked potentials (AEPs) and mood in humans. *Neuropsychobiology* 20:145–51.

BOWLBY, J. 1969. *Attachment.* New York: Basic Books.

———. 1973. *Separation: Anxiety and anger.* New York: Basic Books.

BREIER, A., M. ALBUS, D. PICKAR, T. P. ZAHN, O. M. WOLKOWITZ, AND S. M. PAUL. 1987. Controllable and uncontrollable stress in humans: Alterations in mood and neuroendocrine and psychophysiological function. *American Journal of Psychiatry* 144:1419–25.

BROWN, D. 1981. General stress in anthopological research. *American Anthropologist* 83:74–91.

———. 1982. Physiological stress and culture change in a group of Filipino-Americans: A preliminary investigation. *Human Biology* 9:553–63.

BROWN, G. L., M. H. EBERT, D. C. GOYER, D. C. JIMERSON, W. J. KLEIN, W. E. BUNNEY, AND F. K. GOODWIN. 1982. Aggression, suicide, and serotonin: Relationships to CSF amine metabolites. *American Journal of Psychiatry* 139:741–46.

BUSS, A. H., AND R. PLOMIN. 1984. *Temperament: Early developing personality traits.* Hillsdale: Lawrence Erlbaum Associates.

CLARKE, A. S. 1993. Social rearing effects on HPA axis activity over early development and in response to stress in rhesus monkeys. *Developmental Psychobiology* 26:433–46.

COE, C. L., G. LUBACH, AND W. B. ERSHLER. 1989. Immuno-

logical consequences of maternal separation in infant primates. *New Directions for Child Development* 45:65–78.
COE, C. L., S. G. WIENER, L. T. ROSENBERG, AND S. LEVINE. 1985. "Endocrine and immune responses to separation and maternal loss in nonhuman primates," in *The psychobiology of attachment and separation.* Edited by M. Reite and T. Field, pp. 163–99. New York: Academic Press.
COHEN, P., C. N. VELEZ, J. BROOK, AND J. SMITH. 1989. Mechanisms of the relation between perinatal problems, early childhood illness, and psychopathology in late childhood and adolescence. *Child Development* 60:701–9.
COHEN, S., D. A. TYRRELL, AND A. P. SMITH. 1991. Psychological stress and susceptibility to the common cold. *New England Journal of Medicine* 325:606–12.
———. 1993. Negative life events, perceived stress, negative effect, and susceptibility to the common cold. *Journal of Personality and Social Psychology* 64:131–40.
DALY, M., AND M. WILSON. 1988. Evolutionary social psychology and family homicide. *Science* 242:519–24.
———. 1995. "Discriminative parental solicitude and the relevance of evolutionary models to the analysis of motivational systems," in *The cognitive neurosciences.* Edited by M. S. Gazzaniga, pp. 1269–86. Cambridge: MIT Press.
DE BELLIS, M., G. P. CHROUSOS, L. D. DORN, L. BURKE, K. HELMERS, M. A. KLING, P. K. TRICKETT, AND F. W. PUTNAM. 1993. Hypothalamic-pituitary-adrenal axis dysregulation in sexually abused girls. *Journal of Clinical Endocrinology and Metabolism* 78:249–55.
DE KLOET, E. R. 1991. Brain corticosteroid receptor balance and homeostatic control. *Frontiers in Neuroendocrinology* 12:95–164.
DUNN, A. J. 1989. Psychoneuroimmunology for the psychoneuroendocrinologist: A review of animal studies of nervous system–immune system interactions. *Psychoneuroendocrinology* 14:251–74.
———. 1995. "Interactions between the nervous system and the immune system: Implications for psychopharmacology," in *Psychopharmacology: The fourth generation of progress.* Edited by F. R. Bloom and D. J. Kupfer. New York: Raven Press.
ELLISON, P. 1988. Human salivary steroids: Methodological considerations and applications in physical anthropology. *Yearbook of Physical Anthropology* 31:115–42.
———. 1994. Advances in human reproductive ecology. *Annual Review of Anthropology* 23:2255–75.
EVANS, P., AND M. PITTS. 1994. Vulnerability to respiratory infection and the four-day desirability dip: Comments on Stone, Porter & Neale (1993). *British Journal of Medical Psychology* 67:387–89.
EVERLY, G. S., AND SOBELMAN, S. A. 1987. *Assessment of the human stress response: Neurological, biochemical, and psychological foundations.* New York: AMS.
FINKELHOR, D., AND J. DZUIBA-LEATHERMAN. 1994. Victimization of children. *American Psychologist* 49:173–83.
FLINN, M. V. 1988. Step and genetic parent/offspring relationships in a Caribbean village. *Ethnology and Sociobiology* 9(3):1–34.
———. 1992. "Paternal care in a Caribbean village," in *Father-child relations: Cultural and biosocial contexts.* Edited by B. Hewlett, pp. 57–84. Hawthorne: N.Y.: Aldine de Gruyter.
FLINN, M. V., S. DECKER, D. TEDESCHI, M. TURNER, R. QUINLAN, C. BAERWALD, AND B. G. ENGLAND. 1995. Life history variation of male hormone profiles in a rural Caribbean village (abstract). *American Journal of Physical Anthropology,* suppl. 15:81.
FLINN, M. V., B. G. ENGLAND, AND T. BEER. 1992. Health condition and corticosteroid stress response among children in a rural Dominican village (abstract). *American Journal of Physical Anthropology,* suppl. 14:73.
FLINN, M. V., M. QUINLAN, R. QUINLAN, M. TURNER, AND B. G. ENGLAND. 1995*a*. Glucocorticoid stress response, immune function, and illness among children in a rural Caribbean village (abstract). *American Journal of Human Biology* 7:122.
FUCHS, E., AND G. FLUGGE. 1995. Modulation of binding sites for corticotropin-releasing hormone by chronic psychosocial stress. *Psychoneuroendocrinology* 30:33–51.
GARMEZY, N. 1983. "Stressors of childhood," in *Stress, coping, and development in children.* Edited by N. Garmezy and M. Rutter, pp. 43–83. New York: McGraw-Hill.
GLASER, R., AND J. K. KIECOLT-GLASER. 1994. Editors. *Handbook of human stress and immunity.* New York: Academic Press.
GOLDBERG, L. R. 1992. The development of markers for the big-five factor structure. *Psychological Assessment* 4:26–42.
———. 1993. The structure of phenotypic personality traits. *American Psychologist* 48:26–34.
GOODMAN, A. H., R. B. THOMAS, A. C. SWEDLUND, AND G. J. ARMELAGOS. 1988. Biocultural perspectives on stress in prehistoric, historical, and contemporary population research. *Yearbook of Physical Anthropology* 31:169–202.
GOTTMAN, J. M., AND L. F. KATZ. 1989. Effects of marital discord on young children's peer interaction and health. *Developmental Psychology* 25:373–81.
GRAY, J. A. 1987. 2d edition. *The psychology of fear and stress.* Cambridge: Cambridge University Press.
GUNNAR, M. 1992. Reactivity of the hypothalamic-pituitary-adrenocortical system to stressors in normal infants and children. *Pediatrics* 90:491–97.
GUNNAR, M., M. C. LARSON, L. HERTSGAARD, M. L. HARRIS, AND L. BRODERSEN. 1992. The stressfulness of separation among nine-month-old infants: Effects of social context variables and infant temperament. *Child Development* 63: 290–303.
HANNA, J. M., G. D. JAMES, AND J. M. MARTZ. 1986. "Hormonal measures of stress," in *The changing Samoans.* Edited by P. T. Baker, J. M. Hanna, and T. S. Baker, pp. 203–21. New York: Oxford Unversity Press.
HERBERT, T. B., AND S. COHEN. 1993. Stress and immunity in humans: A meta-analytic review. *Psychosomatic Medicine* 55:364–79.
HEWLETT, B. 1992. "Husband-wife reciprocity and the father-infant relationship among Aka pygmies," in *Father-child relations: Cultural and biosocial contexts.* Edited by B. Hewlett, pp. 57–84. Hawthorne: N.Y.: Aldine.
HIRAMATSU, R. 1981. Direct assay of cortisol in human saliva by solid phase radioimmunoassay and its clinical applications. *Clinica Chimica Acta* 117:239–49.
HOLL, R., H. FEHM, K. VOIGT, AND W. TELLER. 1984. The "mid-day surge" in plasma cortisol induced by mental stress. *Hormones and Metabolism Research* 16:158–59.
HOUSE, J. S., K. R. LANDIS, AND D. UMBERSON. 1988. Social relationships and health. *Science* 241:540–44.
HUBERT, W., AND R. DE JONG-MEYER. 1989. Emotional stress and saliva cortisol response. *Journal of Clinical Chemistry and Clinical Biochemistry* 27:235–37.
HURTADO, A. M., AND K. R. HILL. 1992. "Paternal effect on offspring survivorship among Ache and Hiwi hunter-gatherers: Implications for modeling pair-bond stability," in *Father-child relations: Cultural and biosocial contexts.* Edited by B. Hewlett. New York: Aldine de Gruyter.
INSEL, T. R. 1990. "Long-term neural consequences of stress during development: Is early experience a form of chemical imprinting?" in *The brain and psychopathology.* Edited by B. T. Carroll. New York: Raven Press.
INSEL, T. R., AND L. SHAPIRO. 1992. *Proceedings of the National Academy of Sciences (U.S.A.)* 89:5981–85.
IVANOVICI, A. M., AND W. J. WIEBE. 1981. "Towards a working 'definition' of 'stress': A review and critique," in *Stress effects on natural ecosystems.* Edited by G. W. Barrett and R. Rosenberg, pp. 13–17. New York: Wiley.
JAMES, G. D., D. E. CREWS, AND J. PEARSON. 1989. "Catecholamines and stress," in *Human population biology: A transdisciplinary science.* Edited by M. A. Little and J. D. Haas, pp. 280–95. Oxford: Oxford University Press.
KAGAN, J. 1984. *The nature of the child.* New York: Basic Books.

KAGAN, J., J. S. RESNICK, AND N. SNIDMAN. 1988. The biological basis of childhood shyness. *Science* 240:167–71.

KAPLAN, H. B. 1991. Social psychology of the immune system: A conceptual framework and review of the literature. *Social Science and Medicine* 33:909–23.

KIRSCHBAUM, C., AND D. H. HELLHAMMER. 1989. Salivary cortisol in psychobiological research: An overview. *Neuropsychobiology* 22:150–69.

———. 1994. Salivary cortisol in psychoneuroendocrine research: Recent developments and applications. *Psychoneuroendocrinology* 19:313–33.

KONNER, M. J. 1981. "Evolution and human behavior development," in *Handbook of cross-cultural human development.* Edited by R. H. Munroe, R. L. Munroe, and B. B. Whiting. New York: Garland Press.

LAMB, M., J. PLECK, E. CHARNOV, AND J. LEVINE. 1987. "A biosocial perspecive on paternal behavior and involvement," in *Parenting across the lifespan: Biosocial dimensions.* Edited by J. B. Lancaster, J. Altmann, A. Rossi, and L. Sherrod, pp. 111–42. Hawthorne, N.Y.: Aldine de Gruyter.

LANCASTER, J., AND C. S. LANCASTER. 1987. "The watershed: Change in parental-investment and family-formation strategies in the course of human evolution," in *Parenting across the lifespan: Biosocial dimensions.* Edited by J. B. Lancaster, J. Altmann, A. S. Rossi, and L. R. Sherrod, pp. 187–206. New York: Aldine de Gruyter.

LANDON, J., D. S. SMITH, L. A. PERRY, AND A. A. K. AL-ANSAY. 1984. "The assay of salivary cortisol," in *Immunoassays of steroids in saliva: Proceedings of the Tenth Tenovous Workshop.* Edited by G. F. Read, D. Riad-Fahmy, R. F. Walker, and K. Griffiths, pp. 300–307. Cardiff: Alpha Omega.

LEVINE, S. 1993. The influence of social factors on the response to stress. *Psychotherapy and Psychosomatics* 60:33–38.

LEVY, F., K. M. KENDRICK, E. B. KEVERNE, V. PIKETTY, AND P. POINDRON. 1992. Intracerebral oxytocin is important for the onset of maternal behavior in inexperienced ewes delivered under peridural anesthesia. *Behavioral Neuroscience* 106: 427–32.

LONG, B., G. UNGPAKORN, AND G. A. HARRISON. 1993. Home-school differences in stress hormone levels in a group of Oxford primary school children. *Journal of Biosocial Science* 25:73–78.

MC BURNETT, K., B. B. LAHEY, P. J. FRICK, C. RISCH, R. LOEBER, E. L. HART, M. A. CHRIST, AND K. S. HANSON. 1991. Anxiety, inhibition, and conduct disorder in children. 2. Relation to salivary cortisol. *Journal of the American Academy of Child and Adolescent Psychiatry* 30:192–96.

MC EWEN, B. S. 1995. "Stressful experience, brain, and emotions: Developmental, genetic, and hormonal influences," in *The cognitive neuroscience.* Edited by M. S. Gazzaniga, pp. 1117–35. Cambridge: MIT Press.

MC GAUGH, J. L. 1989. Involvement of hormonal and neuromodulatory systems in the regulation of memory storage. *Annual Review of Neurosciences* 12:255–87.

MAGNANO, C. L., J. M. GARDNER, AND B. Z. KARMEL. 1992. Differences in salivary cortisol levels in cocaine-exposed and noncocaine-exposed NICU infants. *Developmental Psychobiology* 25:93–103.

MAIER, S. F., L. R. WATKINS, AND M. FLESHNER. 1994. Psychoneuroimmunology: The interface between behavior, brain, and immunity. *American Psychologist* 49:1004–17.

MARTIGNONI, E., A. COSTA, E. SINFORIANI, A. LUZZI, P. CHIODINI, M. MAURI, G. BONO, AND G. NAPPI. 1992. The brain as a target for adrenocortical steroids: Cognitive implications. *Psychoneuroendocrinology* 17:343–54.

MARTINEZ, J. L. 1986. "Memory: Drugs and hormones," in *Learning and memory: A biological view.* Edited by J. L. Martinez and R. P. Kesner, pp. 127–63. New York: Academic Press.

MASON, J. W. 1968. A review of psychoendocrine research on the pituitary-adrenal cortical system. *Psychosomatic Medicine* 30:576–607.

———. 1971. A re-evaluation of the concept of "non-specificity" in stress theory. *Journal of Psychosomatic Research* 8:323–34.

MASON, J. W., E. L. BUESCHER, M. L. BELFER, M. S. ARTENSTEIN, AND E. H. MOUGEY. 1979. A prospective study of corticosteroids and catecholamine levels in relation to viral respiratory illness. *Journal of Human Stress,* September, pp. 18–28.

MEANEY, M., J. MITCHELL, D. AITKEN, BHAT AGAR, S. BODNOFF, L. IVY, AND A. SARRIEV. 1991. The effects of neonatal handling on the development of the adrenocortical response to stress: Implications for neuropathology and cognitive deficits later in life. *Psychoneoroendocrinology* 16:85–103.

MONTAGER, H., J. C. HENRY, M. LOMBARDOT, A. RESTOIN, D. BOLZONI, M. DURAND, Y. HUMBERT, AND A. MOYSE. 1978*a*. "Behavioural profiles and corticosteroid excretion rhythms in young children, part 1. Non-verbal communication and setting up of behavioural profiles in children from 1 to 6 years," in *Human behaviour and adaptation.* Edited by V. Reynolds and N. Blurton-Jones. London: Taylor and Francis.

MONTAGER, H., J. C. HENRY, M. LOMBARDOT, M. BENEDINI, J. BURNOD, AND R. M. NICOLAS. 1978*b*. "Behavioural profiles and corticosteroid excretion rhythms in young children, part 2. Circadian and weekly rhythms in corticosteroid excretion levels of children as indicators of adaptation to social context," in *Human behaviour and adaptation.* Edited by V. Reynolds and N. Blurton-Jones. London: Taylor and Francis.

MONTAGER, H., A. RESTOIN, AND J. C. HENRY. 1982. "Biological defense rhythms, stress, and communication in children," in *Reviews of child development research.* Edited by W. Hartup and H. Pick, pp. 291–319. Chicago: University of Chicago Press.

MUNCK, A., AND P. M. GUYRE. 1991. "Glucocorticoids and immune function," in *Psychoneuroimmunology.* Edited by R. Ader, D. L. Felten, and N. Cohen. San Diego: Academic Press.

MUNCK, A., P. M. GUYRE, AND N. J. HOLBROOK. 1984. Physiological functions of glucocorticoids in stress and their relation to pharmacological actions. *Endocrine Reviews* 5:25–44.

NEWCOMER, J. W., S. CRAFT, T. HERSHEY, K. ASKINS, AND M. E. BARDGETT. 1994. Glucocorticoid-induced impairment in declarative memory performance in adult humans. *Journal of Neuroscience* 14:2047–53.

PEARSON, J. D., G. D. JAMES, AND D. E. BROWN. 1993. Stress and changing lifestyles in the Pacific: Physiological stress responses of Samoans in rural and urban settings. *American Journal of Human Biology* 5:49–60.

PETROVICH, S. B., AND J. L. GEWIRTZ. 1985. "The attachment learning process and its relation to cultural and biological evolution: Proximate and ultimate considerations," in *The psychobiology of attachment and separation.* Edited by M. Reite and T. Fields, pp. 259–91. New York: Academic Press.

QUINLAN, R. n.d. Time allocation to parental investment in a Dominican village. Master's thesis, University of Missouri, Columbia, Mo.

RIAD-FAHMY, D., G. F. READ, AND I. A. HUGHES. 1983. "Corticosteroids," in *Hormones in the blood.* Edited by C. H. Gray and V. H. T. James, pp. 285–315. New York: Academic Press.

ROSE, R. M. 1980. Endocrine responses to stressful psychological events. *Advances in Psychoneuroendocrinology: Psychiatric Clinics of North America* 3:251–76.

SAPHIER, D., J. E. WELCH, G. E. FARRAR, N. Q. NGUNEN, F. AGUADO, T. R. THALLER, AND D. S. KNIGHT. 1994. Interactions between serotonin, thyrotropin-releasing hormone, and substance P in the CNS regulation of adrenocortical secretion. *Psychoneuroendocrinology* 19:779–97.

SAPOLSKY, R. M. 1986. Glucocorticoid toxicity in the hippocampus: Reversal by supplementation with brain fuels. *Journal of Neuroscience* 6:2240–45.

———. 1991. "Effects of stress and glucocorticoids on hippocampal neuronal survival," in *Stress: Neurobiology and neuroendocrinology.* Edited by M. R. Brown, G. F. Koob, and C. Rivier, pp. 293–322. New York: Dekker.

———. 1992*a*. "Neuroendocrinology of the stress-response," in *Behavioral endocrinology.* Edited by J. B. Becker, S. M. Breedlove, and D. Crews, pp. 287–324. Cambridge: MIT Press.

———. 1994. *Why zebras don't get ulcers.* New York: Freeman.

SCHEPER-HUGHES, N. Editor. 1988. *Child survival: Anthropo-*

logical perspectives on the treatment and maltreatment of children. Boston: Reidel.
SCHNEIDER, M. L., C. L. COE, AND G. R. LUBACH. 1992. Endocrine activation mimics the adverse effects of prenatal stress on the neuromotor development of the infant primate. *Developmental Psychobiology* 25:427–39.
SEYLE, H. 1976. Revised edition. *The stress of life.* New York: McGraw-Hill.
TENNES, K., AND J. MASON. 1982. "Developmental psychoendocrinology: An approach to the study of emotions," in *Measuring emotions in infants and children.* Edited by C. Izard, pp. 21–37. London: Cambridge University Press.
TINBERGEN, N. 1974. Ethology and stress diseases. *Science* 185:20–27.
TURNER, M. T. n.d. Mother and infant cortisol response in a Dominican village. Master's thesis, University of Missouri, Columbia, Mo.
TURNER, M. T., M. V. FLINN, AND B. G. ENGLAND. 1995. Mother-infant glucocorticoid stress response in a rural Caribbean village. *American Journal of Physical Anthropology,* suppl. 15:94.
UMEDA, T., R. HIRAMATSU, T. IWAOKA, T. SHIMADA, F. MIURA, AND T. SATO. 1981. Use of saliva for monitoring unbound free cortisol levels in serum. *Clinica Chimica Acta* 110:245–53.
VAN CAUTER, E. 1990. Diurnal and ultradian rhythms in human endocrine function: A mini-review. *Hormone Research* 34:45–53.
VAN WIMERSMA GREIDANUS, TJ. B., AND D. DE WIED. 1977. "The physiology of the neurohypophyseal system and its relation to memory processes," in *Biochemical correlates of brain structure and function.* Edited by A. M. Davison, pp. 215–48. New York: Academic Press.
VINING, R. F., R. A. MC GINLEY, J. J. MAKSVYTIS, AND K. Y. HO. 1983. Salivary cortisol: A better measure of adrenal cortical function than serum cortisol. *Annals of Clinical Biochemistry* 20:329–35.
WEISNER, T. S. 1981. "Cities, stress, and children: A review of some cross-cultural questions," in *Handbook of cross-cultural human development.* Edited by R. H. Munroe, R. L. Munroe, and B. B. Whiting. New York: Garland Press.
WEISNER, T. S., AND S. ABBOTT. 1977. Women, modernity, and stress: Three contrasting contests for change in East Africa. *Journal of Anthropological Research* 33:421–51.
WERNER, E. E. 1985. "Stress and protective factors in children's lives," in *Longitudinal studies in child psychology and psychiatry.* Edited by A. R. Nicol. New York: Wiley.
WILSON, M. I., M. DALY, AND S. J. WEGHORST. 1980. Household composition and the risk of child abuse and neglect. *Journal of Biosocial Science* 12:333–40.
WINSLOW, J. T., N. HASTINGS, C. S. CARTER, C. R. HARBAUGH, AND T. R. INSEL. 1993. A role for central vasopressin in pair bonding in monogamous prairie voles. *Nature* 365:545–48.
WOLKOWITZ, O. M., V. I. REUS, H. WEINGARTNER, K. THOMPSON, A. BREIER, A. DORAN, D. RUBINOW, AND D. PICKAR. 1990. Cognitive effects of corticosteroids. *American Journal of Psychiatry* 147:1297–1303.
YEHUDA, R., E. L. GILLER, S. M. SOUTHWICK, M. T. LOWY, AND J. W. MASON. 1991. Hypothalamic-pituitary-adrenal dysfunction in posttraumatic stress disorder. *Biological Psychiatry* 30:1031–48.

Between Two Poles: Bronislaw Malinowski, Ludwik Fleck, and the Anthropology of Science[1]

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Exploring the public impact of popular anthropological writing, we began with the idea that Malinowski had not been properly credited with influencing the contemporary science-studies movement with his popular essay "Magic, Science, and Religion," a work that was widely read and circulated after its appearance in a volume on science and religion edited by Joseph Needham (1925). As we pursued this idea, we came to recognize a parallel effort by another scholar of Polish origin to challenge the notion of a "prelogical" human mentality. What follows, therefore, is a note on the history of ideas and the part played by anthropologists in that history.

Bronislaw Malinowski and Ludwik Fleck were born in the last few decades of the 19th century in the part of Austria-Hungary that would eventually become Poland. Both were trained in the natural sciences but also well-read in philosophy and the social sciences. Both opposed Lévy-Bruhl's (1923) idea of a "prelogical" mentality (Fleck 1979[1935]:155; Malinowski 1948[1925]:25–26). Finally, both relied on extensive field experience—Malinowski in the Trobriands and Fleck in the medical laboratories and hospitals of Lvov—to make critical points about scientific knowledge. Malinowski's definitive statement on fieldwork and its methodology is widely read and need not be repeated here. Less well-known is the significance of participant observation in Fleck's research (Lowy 1988:148–49):

> [Fleck's] professional experience allowed him to use concrete and detailed examples from the daily practice of a clinic, a hospital, a medical analysis laboratory. . . . His viewpoint was simultaneously an "inside" and an "outside" one. It allowed him to combine the advantages of an internal participant with those of an external observer. His specific position—a fundamental scientist in a temporary (or so he hoped) "exile" among practitioners—made it easier for him to observe the shortcomings and the difficulties of the application of science to medicine.

In short, both men were participant observers able to juxtapose two worlds and through this anthropological juxtaposition to launch their respective critiques of Western science.

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