Invasive Fungal Dermatitis in the ≤1000-Gram Neonate
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Invasive Fungal Dermatitis in the ≤1000-Gram Neonate

Judith L. Rowen, MD*; Jane T. Atkins, MD†; Moise L. Levy, MD§; Susan C. Baer, MD||, and Carol J. Baker, MD*

ABSTRACT. Objective. In 1991, we noted the emergence amongst our extremely low birth weight neonates of a new clinical entity, invasive fungal dermatitis, characterized by erosive, crusting lesions and a high rate of subsequent systemic fungal infection. We sought to define this condition and examine potential risk factors.

Methods. Sixteen neonates with invasive fungal dermatitis were seen during a 2-year period in three Baylor College of Medicine affiliated intensive care nurseries. Seven were confirmed cases, with skin biopsy evidence of invasion beyond the stratum corneum. Nine had a consistent clinical course and a positive potassium hydroxide examination of skin scrapings or isolation of fungi from skin or systemic cultures. Three controls were matched to each case by hospital, date of admission, and birth weight. Data was collected by retrospective chart review.

Results. Invasive fungal dermatitis occurred in 5.9% of at-risk infants. Case patients had a mean birth weight of 635 g and developed skin lesions at a mean age of 9 days (range, 6 to 14). Candida albicans was the most commonly implicated pathogen, but other Candida species, Aspergillus, Trichosporon beigellii, and Curvularia were also seen. Disseminated infection occurred in 69%, all due to Candida sp. Case patients were significantly more premature than controls (mean gestation, 24.4 vs 25.9 weeks) and were more likely to be delivered vaginally (81% vs 50%). Postnatal steroids were administered to cases (81%) more often than controls (46%). Case patients had more prolonged hyperglycemia (as assessed by insulin administration) than controls (mean 4.3 vs 2.0 days).

Conclusions. Invasive fungal dermatitis is a disease of the smallest, most immature neonates and is associated with vaginal birth, steroid administration, and hyperglycemia. We speculate that the skin serves as a portal of entry for colonizing fungal species and may thus lead to disseminated infection. Methods to improve skin barrier function may be useful in preventing this disorder. Pediatrics 1995; 95:682–687; invasive fungal dermatitis, lesions, neonates, skin, Candida, Aspergillus, Trichosporon, Curvularia.

ABBREVIATION. KOH, potassium hydroxide.

As the survival of infants with birth weights ≤1500 g has improved, a new spectrum of infectious agents associated with these susceptible hosts has emerged. In addition to the usual neonatal pathogens, these infants develop infections due to nosocomially acquired commensals such as coagulase negative staphylococci and fungi.1 Systemic fungal infection due to Candida occurs in 1.6 to 5% of these infants.2–5 Other fungi such as Malassezia, Aspergillus, and Trichosporon are described as pathogens in premature infants albeit their occurrence is rare.6–8 Intravascular catheters are often the presumed portal of entry for these fungi but Candida and Aspergillus may also disseminate after their overgrowth in the gastrointestinal tract.27 Once dissemination occurs, nearly any organ can be involved.26–8

Cutaneous involvement occurs in 50 to 60% of very low birth weight neonates with systemic candidiasis and in 29% of those with aspergillosis.27 Dermatologic findings in candidiasis include diffuse erythematous rash, diaper dermatitis, or skin abscesses, often at the site of intravascular catheters.29 Congenital candidiasis is characterized by an extensive skin rash, usually within 12 hours of birth. This often resolves spontaneously in term infants, but may proceed to invasive disease and death in very low birth weight infants.10,11 In 1991 we recognized what appeared to be a new expression of cutaneous fungal infection in ≤1000 g neonates. Skin involvement was characterized by ulcerative and erosive lesions with extensive crusting. Unlike congenital candidiasis, these lesions did not appear until the infant was several days old rather than being present at birth. These cutaneous findings often were associated with systemic involvement. We propose that this entity, invasive fungal dermatitis, represents an alternative portal of entry in the development of systemic fungal infection among extremely low birth weight neonates.

METHODS

Cases. Sixteen neonates with a diagnosis of invasive fungal dermatitis were cared for at the three Baylor College of Medicine affiliated hospital nurseries in the 2-year period from June 1991 through May 1993. One of these patients has been reported previously.7 All patients were evaluated by the Infectious Diseases or Dermatology consultation services. Cases were classified as confirmed if the infant had a classic appearance of black, white, or buff crusts on the skin, often with ulceration and eschar, and a skin biopsy demonstrating invasion beyond the stratum corneum. Cases were categorized as probable if the above dermatologic features were present and the patient had a positive KOH (potassium hydroxide) preparation of skin scrapings or a positive systemic culture or a surface culture from a skin lesion that grew...
During the 2-year study period, 271 neonates with birth weights ≤1000 g survived to age 6 days (the Fig 1. Photograph of patient 16 (see case report). Diffuse involvement of the back and buttocks with tightly adherent white and buff colored crust. Small areas of ulceration are evident.

Controls. Three controls were selected for each case. The controls were matched for the hospital and date of admission. They were also matched within two birth weight groups, either ≤750 g or 751 to 1000 g. Controls were accepted only if they survived at least until the age of diagnosis for the corresponding matched case. Two potential controls were replaced. One was replaced because the chart could not be located; another because the infant had a 5-cm wide, 2-cm deep sacral lesion that appeared black and "fungus-like," but no cultures were obtained and the infant died shortly thereafter. Chart review for control infants was limited to collection of the following: birth weight, gestational age, sex, final outcome (discharge or death), mode of delivery, steroid administration, days of antibiotic use, presence of hyperglycemia (as gauged by insulin administration), and use of occlusive dressings. For cases and matched controls, charts were reviewed through the day of diagnosis.

Statistical Methods. Comparisons between cases and controls were made using the $X^2$ with Yates correction, Fisher's exact test, or unpaired two-tailed $t$ test, as appropriate.

Illustrative Case

This 570-g birth weight, 25 weeks' gestation male infant was born via precipitous footling breech vaginal delivery complicated by head entrapment. Apgar scores were 2 at 1 minute, 5 at 5 minutes, and 6 at 10 minutes. The mother received 48 hours of parenteral ampicillin before delivery. The infant was intubated, umbilical arterial and venous catheters were placed, and ampicillin, gentamicin, Exosurf and intravenous immunoglobulin were administered. The infant was sedated with morphine and phenobarbital; his head and extremities were swathed in plastic wrap and Tegaderm was placed over his back and chest to aid thermo-regulation. Serum glucose levels near 300 were treated with insulin on several occasions beginning on day 2 of life. Hyperbilirubinemia necessitated an exchange transfusion. That evening, the infant had an acute respiratory decompensation. One day 3 of life, a pustule was noted on the infant's right arm, the plastic wrap was removed and the area dried. On day 6 of life, a head ultrasound revealed a grade IV intraventricular hemorrhage on the right, with grade III on the left. A patent ductus arteriosus was surgically ligated on day 7. Caffeine therapy was begun. The umbilical venous catheter was removed.

On day 8, the skin of the patient's left groin and penis were noted to be macerated, friable, and erythematous and the areas that had been covered by Tegaderm had crusty, whitish plaques (see Fig 1). KOH preparation of scrapings from the plaques revealed hyphae. Cultures were obtained and amphotericin B and topical antifungal therapy were instituted. Cultures of the lesions grew Candida albicans, but blood and urine cultures were sterile. The umbilical artery catheter was removed on day 11 of life. His condition deteriorated on day 12 of life, necessitating therapy with vancomycin, amikacin, and dopamine. Blood cultures obtained on days 10, 13, and 14 of life grew C albicans. An abdominal ultrasound revealed a distended gallbladder but no evidence of renal, hepatic or splenic fungal infection. The patient developed necrotizing enterocolitis; clindamycin was added to therapy. Exploratory laparotomy on day 15 of life revealed jejunal perforation with fecal peritonitis; peritoneal fluid grew C albicans. The patient received a total of 30 mg/kg of amphotericin B, with apparent resolution of his systemic candidiasis. He died at 108 days of age without evidence of candidal infection by postmortem examination.

RESULTS

During the 2-year study period, 271 neonates with birth weights ≤1000 g survived to age 6 days (the
youngest age at diagnosis for case patients). The incidence of invasive fungal dermatitis was 5.9% in this at-risk population. There was no seasonal predilection. Clinical characteristics of our sixteen cases are summarized in Table 1. Seven were biopsy confirmed, and nine were classified as probable. Mean birth weight was 635 g and mean gestational age was 24.4 weeks. Invasive fungal dermatitis occurred at a mean age of 9 days (range 6 to 14). Eleven (69%) of the infections were caused by C albicans. The other five were associated with C tropicalis, C parapsilosis and Aspergillus niger, A fumigatus, Trichosporon beigelli, and Curvularia sp.

Evidence of disseminated infection was found in 11 patients (69%), all associated with Candida sp. Manifestations of systemic infection included fungemia (7), meningitis (4), urinary tract infection (2), and peritonitis (1). Patient 6 had postmortem evidence of renal and hepatosplenic involvement. In four of these infants, recognition of the skin lesions provoked collection of the positive systemic cultures. Five patients grew yeast from systemic cultures obtained 1 to 4 days after recognition of skin lesions. Two other patients with disseminated infections had systemic cultures collected 1 or 2 days before diagnosis of invasive fungal dermatitis.

Three of the seven skin biopsy specimens (patients 1, 2, and 7) revealed invasion of fungi into the dermis. The remaining four had invasion by fungal elements restricted to epidermal structures, usually with obvious inflammatory infiltrate within the dermis. The infant infected with Curvularia had some granuloma formation in the dermis. Focal necrosis and hemorrhage were noted in several biopsy specimens. Representative biopsies are depicted in Fig 2.

A summary of the results of the risk factor analysis from the case control study is found in Table 2. Due to sample size considerations, multivariate analysis could not be performed and it is possible some of the identified risk factors are linked. Although the patients and controls were matched for birth weight, controls had a significantly greater gestation (mean, 25.9 weeks) when compared with cases (mean, 24.4 weeks). Affected neonates were more likely to be delivered vaginally (61%) than controls (50%). Steroids, usually a single dose of intravenous dexamethasone to improve refractory hypotension, were administered to 81% of cases but to only 46% of controls. Infected infants frequently had prolonged hyperglycemia requiring insulin therapy; cases required insulin for a mean of 4.3 days compared with 2.0 for controls. Insulin therapy was instituted at the discretion of the attending physician; dosing was usually based on serum glucose levels but occasionally was prompted by glucosuria. Although antibiotics were administered longer and occlusive dressings were applied directly to the skin more often in infected infants than controls, neither were statistically significant. Analysis of occlusive dressing use still showed no difference between cases and controls when limited to nonsterile (eg, plastic wrap) products. Patients and controls also did not differ in survival rate.

**DISCUSSION**

Our 16 neonates with invasive fungal dermatitis comprise the largest series and the first report of skin

### TABLE 1. Case Summaries

<table>
<thead>
<tr>
<th>Patient</th>
<th>BW, g</th>
<th>EGA, wk</th>
<th>Age, d</th>
<th>Fungal Species</th>
<th>Method of Dx</th>
<th>Systemic Disease</th>
<th>Treatment (mg/kg) Amphotericin B</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Confirmed Cases</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>690</td>
<td>24.6</td>
<td>14</td>
<td>C albicans</td>
<td>Biopsy</td>
<td>Fungemia</td>
<td>30</td>
<td>Residual scarring</td>
</tr>
<tr>
<td>2</td>
<td>820</td>
<td>24</td>
<td>7</td>
<td>C albicans</td>
<td>Biopsy</td>
<td>Fungemia</td>
<td>33</td>
<td>Recovered</td>
</tr>
<tr>
<td>3</td>
<td>855</td>
<td>29</td>
<td>9</td>
<td>C albicans</td>
<td>Biopsy</td>
<td>Meningitis</td>
<td>25.5</td>
<td>Recovered</td>
</tr>
<tr>
<td>4</td>
<td>509</td>
<td>23.1</td>
<td>8</td>
<td>T beigelli</td>
<td>Biopsy</td>
<td>Unknown</td>
<td>3.4</td>
<td>Died, apparent sepsis</td>
</tr>
<tr>
<td>5</td>
<td>510</td>
<td>23.1</td>
<td>8</td>
<td>Curvularia sp.</td>
<td>Biopsy</td>
<td>None</td>
<td>25.5</td>
<td>Died, NEC</td>
</tr>
<tr>
<td>6</td>
<td>615</td>
<td>25</td>
<td>7</td>
<td>C parapsilosis; A niger</td>
<td>Biopsy</td>
<td>UTI</td>
<td>40.5</td>
<td>Recovered</td>
</tr>
<tr>
<td>7</td>
<td>785</td>
<td>25</td>
<td>7</td>
<td>A fumigatus</td>
<td>Biopsy</td>
<td>None</td>
<td>60</td>
<td>Residual scarring</td>
</tr>
<tr>
<td><strong>Probable Cases</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>680</td>
<td>24</td>
<td>10</td>
<td>C albicans</td>
<td>Blood culture</td>
<td>Fungemia, UTI</td>
<td>30.5</td>
<td>Skin healed; died other causes</td>
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<tr>
<td>9</td>
<td>475</td>
<td>22.6</td>
<td>13</td>
<td>C albicans</td>
<td>KOH; blood culture</td>
<td>Fungemia</td>
<td>6.6</td>
<td>Died</td>
</tr>
<tr>
<td>10</td>
<td>605</td>
<td>24</td>
<td>10</td>
<td>C albicans</td>
<td>KOH; skin culture; autopsy</td>
<td>None</td>
<td>30.5</td>
<td>Recovered</td>
</tr>
<tr>
<td>11</td>
<td>640</td>
<td>26.3</td>
<td>8</td>
<td>C albicans</td>
<td>KOH; skin culture; autopsy</td>
<td>Meningitis, renal and hepatosplenic</td>
<td>4.5</td>
<td>Died, MRSA and fungal sepsis</td>
</tr>
<tr>
<td>12</td>
<td>605</td>
<td>24.3</td>
<td>7</td>
<td>C albicans</td>
<td>CSF culture</td>
<td>Meningitis</td>
<td>34.5</td>
<td>Recovered</td>
</tr>
<tr>
<td>13</td>
<td>575</td>
<td>23</td>
<td>11</td>
<td>C albicans</td>
<td>KOH; skin culture</td>
<td>Fungemia, meningitis</td>
<td>4</td>
<td>Died</td>
</tr>
<tr>
<td>14</td>
<td>680</td>
<td>24.3</td>
<td>11</td>
<td>C albicans</td>
<td>KOH; skin culture</td>
<td>Fungemia, meningitis</td>
<td>30</td>
<td>Recovered</td>
</tr>
<tr>
<td>15</td>
<td>545</td>
<td>24.3</td>
<td>9</td>
<td>C tropicalis</td>
<td>Skin and blood cultures</td>
<td>Fungemia</td>
<td>30</td>
<td>Recovered</td>
</tr>
<tr>
<td>16</td>
<td>570</td>
<td>25</td>
<td>9</td>
<td>C albicans</td>
<td>KOH; skin, blood and peritoneal cultures</td>
<td>Fungemia, NEC</td>
<td>30</td>
<td>Died, other causes</td>
</tr>
</tbody>
</table>
Fig 2. Skin biopsy specimens. A, Yeast forms restricted to the stratum corneum (arrows). This infection is not invasive and can be treated with topical therapy. Hematoxylin and eosin, 40 x. B, Abscesses beneath stratum corneum (arrows) in specimen from patient 3. Hematoxylin and eosin, 10 x. C, Intense involvement of epidermis with pseudohyphae extending along hair follicles (arrows). Specimen from patient 2. Grocott-methenamine silver, 10 x. D, Higher magnification of specimen from patient 2, with yeast forms present in dermis (arrows). Grocott-methenamine silver, 40 x.

TABLE 2. Risk Factor Analysis

<table>
<thead>
<tr>
<th></th>
<th>Cases (16)</th>
<th>Controls (48)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>BW (g)*</td>
<td>635 ± 28</td>
<td>690 ± 15</td>
<td>NS</td>
</tr>
<tr>
<td>Sex (proportion male)</td>
<td>56%</td>
<td>58%</td>
<td>NS</td>
</tr>
<tr>
<td>EGA (wk)*</td>
<td>24.4 ± 4</td>
<td>25.9 ± 3</td>
<td>.01</td>
</tr>
<tr>
<td>Vaginal birth</td>
<td>81%</td>
<td>50%</td>
<td>.03</td>
</tr>
<tr>
<td>Steroid days*</td>
<td>81%</td>
<td>46%</td>
<td>.01</td>
</tr>
<tr>
<td>Insulin days*</td>
<td>4.3 ± .7</td>
<td>2.0 ± .4</td>
<td>.006</td>
</tr>
<tr>
<td>Antibiotic days*</td>
<td>8.3 ± .5</td>
<td>6.8 ± .4</td>
<td>NS</td>
</tr>
<tr>
<td>Occlusive dressings</td>
<td>81%</td>
<td>59%</td>
<td>NS</td>
</tr>
<tr>
<td>Survival to discharge</td>
<td>56%</td>
<td>77%</td>
<td>NS</td>
</tr>
</tbody>
</table>

BW, birth weight; EGA, estimated gestational age; NS, not significant, P > .05.

*Mean ± SE.

biopsy findings in these neonates. In their review of cutaneous manifestations of systemic candidiasis in neonates, Baley and Silverman9 do not describe patients with similar erosive, crusting lesions. One 7-day-old neonate described during an outbreak of *Trichosporon beigeli* infection had skin breakdown and white plaques.8 In another report a 6-day-old neonate who grew *Aspergillus fumigatus* from epidermal ulcerations is described.12

Although speculative, we believe that the skin is the portal of entry in these neonates and serves as the source for subsequent dissemination of fungal infection. The combined circumstances of early fungal colonization of skin and its immature barrier function in extremely premature infants is compounded by local disruption, allowing invasion and subsequent systemic infection. This early colonization is aided by passage through the birth canal. *Candida* is found in the vagina of 35 to 46% of pregnant women13,14 and two prospective studies have associated vaginal delivery with infant colonization.5,15 *Trichosporon beigeli*, the causative agent of white piedra, has been cultured from the genital area of up to 14% of women, so it is conceivable that vaginal delivery also was a mode of colonization in patient 4.16,17 The source of *Aspergillus* and *Curvularia* in patients 5 to 7 is unclear;
infections caused by these organisms are presumed to arise from environmental sources, but *Aspergillus* has been isolated from vaginal secretions.\(^3\)\(^,\)\(^4\)

Despite being matched by birth weight with controls, our neonates with invasive fungal dermatitis were significantly more premature (mean gestation, 24.4 weeks). With this degree of prematurity, the barrier function of the skin is compromised\(^2\)\(^,\)\(^3\)\(^,\)\(^5\) with reduced epidermal cell numbers and epidermal thickness, as well as immaturity of the dermoeidermal undulations.\(^2\)\(^,\)\(^5\) By 2 postnatal weeks, even the most premature infants achieve epidermal characteristics indistinguishable from those present at birth in term infants.\(^2\)\(^,\)\(^5\) This may explain why none of our patients was older than 14 days.

Many of the procedures performed in the intensive care nursery disrupt the neonate's fragile skin. Tape and monitoring electrodes lead to trauma associated with an increase in transepidermal water loss.\(^2\)\(^,\)\(^6\) A reluctance to manipulate these very ill infants may lead to prolonged positioning, and thus an opportunity for further skin damage. Also, our patients were more likely than controls to have prolonged hyperglycemia, with attendant glucosuria; pooling of such glucose-rich urine could promote growth of fungi colonizing the skin as well as skin maceration. A glucose-enriched environment in vitro enhances adherence of *Candida*,\(^2\)\(^,\)\(^7\) a necessary prelude to dissemination in the pathogenesis of systemic candidiasis. It is also possible that hyperglycemia resulted from fungal infection rather than acting as a contributing factor, as neonates with systemic candidiasis often have carbohydrate intolerance.\(^2\)\(^,\)\(^2\)\(^,\)\(^4\)

Another factor more common in the cases than controls was postnatal use of steroids. We previously reported that steroid use in very low birth weight neonates is significantly associated with *Candida* colonization.\(^2\) Steroid use also has been implicated as a risk factor for disseminated candidiasis.\(^2\)\(^,\)\(^5\) *C. albicans* has receptors for corticosteroids,\(^2\)\(^6\) so there may be direct effects on the fungi. As with glucose, exposure of *Candida* to steroids in vitro increases expression of an adherence molecule on the yeast.\(^2\)\(^,\)\(^7\) Administration of steroids also may disturb local host defenses, thus allowing invasion. Steroid use often induces hyperglycemia; our sample size precludes multivariate analysis to determine whether these two identified risk factors are independent or linked. As the use of steroids for hypotension remains experimental, perhaps our findings should sound a note of caution against their indiscriminate use.

Once the skin barrier is broached, dissemination may occur. In the majority of our patients, skin findings occurred before evidence of systemic infection. The biopsies clearly demonstrate a progression of invasion from the epidermal surface into the dermis. This contrasts with the intact epidermis and extensive dermal disease noted in skin lesions resulting from hematogenous dissemination.\(^2\)\(^8\) Dissemination did not occur in all of our patients, sometimes despite deep cutaneous lesions. Thus, the timing and the variability in systemic findings lead us to conclude that the skin is a primary source of infection in these cases rather than a secondary site.

As with many infections, a high index of suspicion will enhance the likelihood of diagnosis. Extensive skin breakdown and crusting in ≤1000-g neonates should be investigated by KOH preparation of skin scrapings. If hyphae or yeast forms are seen, a biopsy should be considered. Once the diagnosis of invasive fungal dermatitis is considered, amphotericin B therapy is initiated. If systemic disease is documented, skin biopsy may not be indicated as therapy will not be altered. In all other circumstances, however, a skin biopsy is helpful. If no invasion beyond the stratum corneum is noted, topical antifungal therapy may be sufficient. Mycostatin cream (Westwood-Squibb, Buffalo, NY) is used in our institution because of its safety in neonates. This preparation is free of preservatives, such as benzyl alcohol, which might pose a threat if absorbed in large quantities. Invasive disease always warrants systemic amphotericin B therapy, given the high rate of dissemination. For non-*Candida* pathogens, the biopsy may be the only source to define the etiology, and thus, the appropriate length of therapy. In one of our patients (patient 7), as a repeat biopsy continued to show fungal elements after 30 mg/kg of amphotericin B had been administered, therapy was extended.

It appears that preventing invasive fungal dermatitis in extremely low birth weight neonates requires measures to improve the barrier function of their skin. Trauma should be minimized, and these neonates should be maintained in a sterile, dry environment if possible. A variety of semipermeable dressings and emollients have been used for this purpose, with a decrease in transepidermal water loss without increased bacterial or fungal colonization.\(^2\)\(^9\) The frequency of risk factors, such as hyperglycemia or steroid use, are not detailed in these studies, and the newborns studied were slightly more mature than our case patients. The use of such dressings remains experimental, and will require study of a larger group of these high-risk infants to accurately gauge safety. Hopefully, continued investigation in this area will address these concerns and an approach to prevention will be constructed.

In summary, we describe 16 patients with invasive fungal dermatitis, often but not exclusively due to *Candida* sp. Factors associated with its development included extreme prematurity, vaginal birth, steroid administration, and hyperglycemia. We propose that these immature infants acquire fungal skin colonization, usually during vaginal delivery, and due to immature barrier function of their skin and events promoting local disruption, invasion by the colonizing fungal species with subsequent dissemination may occur. Further research into effective intervention to improve skin barrier function may provide a means of preventing this condition.

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HOW TO IMPROVE PUBLIC HEALTH

Doctors and other health workers are even less able to assure happiness than they are to assure health. If work is unsatisfying to many in modern society, psychopharmacology is a toxic and inappropriate remedy for correcting the resulting tension and alienation. If unemployed workers are depressed, mental-health counseling may be a temporary source of comfort, but the only genuine solution is full employment. If children fail to thrive, child-guidance workers may diminish their misery, but they cannot guarantee their flowering in the midst of social disaster. As physicians, our daily practice with human ailments makes us aware of the extent to which problems of ill health flow from failures in our political, economic, and social institutions. The redesign of these institutions is the central challenge for the coming century, and gives the greatest promise for improving public health.

REFERENCE


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