

THE PHOENIX HAS RISEN BUT HAS FAILED TO THRIVE: HOPE ON THE HORIZON FOR KING-DREW MEDICAL CENTER

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History has taught us that sociopolitical trends evolving in the state of California have found their way into the national mainstream. So it is with great trepidation that we witness the systematic dismantling of an historically black- and Hispanic-serving institution whose mission is to train physicians to care for the underserved minority communities of our nation at a time when health disparities among minority populations continue to plague our nation. King-Drew Medical Center is an institution that has served millions of people, saved tens of thousands of lives over several decades, and has trained thousands of physicians who have demonstrated a commitment to its mission. Yet, its history and its current status are as complex as the health disparities experienced by the poor that it was meant to address and end.

A phoenix burned itself to ashes in a pyre of hate and discontent in 1964. For many years, the phoenix, an African-American community—known as Watts, Los Angeles—tolerated racism, social injustice, poverty, and extremely poor access to healthcare. It was only a matter of time before the burning, but, as the legend goes, its resurrection and new life was imminent. The new Watts was born from the passage of civil rights laws and the establishment of the

Charles R. Drew Postgraduate Medical School in 1966—later named the Charles R. Drew University of Medicine and Science (Drew)—with the addition of a College of Allied Health. The establishment of the medical school was promulgated by the findings of the gubernatorial-appointed McCone Commission, which cited lack of access to healthcare as central to fomenting civil discontent.

In 1972, the Martin Luther King General Hospital was constructed across the street from the medical school during the latter's early years. Together, they were designated the King-Drew Medical Center (KDMC), located in an unincorporated region in south Los Angeles primarily serving residents from Compton, Willowbrook, South Central, Lynwood, and Watts. Importantly, it was the first traditionally black medical institution of its kind established west of the Mississippi River. Though it was born to live, grow, succeed, and serve its surrounding community, its failure to thrive began almost immediately.

Metaphorically, KDMC's failure to thrive is not unlike one of the most important unresolved public health problems faced by the community it serves—that of a young, undereducated, pregnant teenager living in poverty. The “infant” that KDMC carried was the mission to train a new breed of physician who would serve the underserved to improve the health status of its community. However, just as is the case with many pregnant youth and their unborn children in south Los Angeles, KDMC faced serious barriers to realizing good outcomes for itself and its mission. It was expected to thrive while in poverty with a neglectful parent—the L.A. County Department of Health Services (LACDHS)—and poor oversight from the responsible agency—the L.A. County Board of Supervisors, a common denominator for poor health outcomes globally.

KDMC's limited funding as a public safety net

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hospital and its limited access to extramural funding and research infrastructure (not being a four-year medical school) have played a large role in the deficiencies that have culminated in struggling residency training programs. This is reminiscent of limited access to medical services, inadequate utilization of prenatal care, and limited educational attainment for many impoverished pregnant teenagers in south Los Angeles. Moreover, the university and the Department of Health Services' historical dependence on bailout solutions to remedy their financial problems compound the problems, which may be traced to ineffective strategic planning and a reluctance to embrace healthcare in the 21st century, as exemplified by L.A. County's reluctance to address civil service system restraints on healthcare delivery and the lack of university initiatives to diversify income streams. Like the poor pregnant teenager who is unemployed, lives on welfare, and engages in substance abuse to forget her problems, KDMC has used quick fixes. Consequently, her problems are never adequately addressed. The result has been a lack of success of the ultimate mission noted, in part, by its service community continuing to have the highest mortality from chronic diseases in L.A. County. KDMC was an unprepared youth in poverty without assurance for good health outcomes for herself and her prematurely born child—both were at risk for premature death from the beginning.

KDMC's circumstance as an institution teaching medical students and training young physicians is unlike that of other medical schools in California. Over time the other two local medical schools—USC and UCLA—in Los Angeles County have developed the ability to generate income from private medical practice activities, private hospital affiliations, private practice faculty plans, large endowments, and separate private medical and hospital facilities. On the other hand, the only historically black- and now-Hispanic serving medical school west of the Mississippi River has an organizational structure that inherently weakens it.

In organizational terms, the university has never developed the capacity to generate private (commercial) sources of revenue that would guarantee its sustainability apart from some limited pharmaceutical sponsorship of its clinical trials unit. Its large dependence on the L.A. County Department of Health Services (LACDHS), which is charged by the L.A. County Board of Supervisors (LACBOS) to allocate public funds for safety-net health servic-

es, is an anomaly in the structure of healthcare and healthcare delivery systems. Ultimately, this puts the fate of the Martin Luther King, Jr. General Hospital and the future of the Charles R. Drew University of Medicine and Science predominantly in the hands of one system that itself is facing tremendous budget deficits and political pressures.

Simultaneously, there are serious concerns regarding the process of care by those in charge of the structure of healthcare for the poor and uninsured in south Los Angeles. For example, LACDHS and LACBOS have been questioned over the triaging of the federal and state financial resources over which they have stewardship. The classical public health model focuses resources on those at greatest risk to ensure they receive at least some minimal healthcare. However, resource allocation is not and has not been commensurate with well-documented morbidity and mortality rates in L.A. County. The worse health outcomes have been and continue to be in the community served by KDMC, and this was the reason for its creation. The lack of confidence from LACDHS and LACBOS in the administration at both the university and the county's own supported hospital have also contributed to limiting resources, which further leads to poor outcomes, a vicious cycle.

KDMC has had the greatest need for resources and serves the community at greatest risk for morbidity and mortality from preventable disease in L.A. County. Therefore, the near-termination of all graduate medical education training and the continued very-poor-health status of south Los Angeles means that the leadership mandate and its attendant fiduciary responsibility to the health of the community have not been met by Drew, LACDHS, and LACBOS. Until each entity recognizes they are in part responsible, KDMC may exist but will not thrive.

At its inception, Martin Luther King General Hospital's organizational culture and medical expertise was community based and only secondarily academic. This meant that patient care at the evolving medical center was meant to be its strength and the other three other components of successful academic medical centers—resource generation, teaching, and research—were nonexistent, or in their infancy. This notwithstanding, over the past 10 years, the revised strategic planning process has led to significant improvements in teaching and research. Drew students must conduct a primary care research project under the mentor-

ing of faculty and oversight of a thesis committee as requisite for graduation. Drew is one of the few medical schools in the nation to make this compulsory. In addition, Drew has focused on the development of a strong research infrastructure concentrating on minority health concerns supported in large part by the National Institutes of Health. This has brought in substantial funding for research, which in the most recent NIH institutional funding report, put Drew ahead of 38 other medical schools with four-year programs in undergraduate medical education. Importantly, Drew ranked 153rd of over 2,800 different research and academic institutions funded by the NIH.

By way of contrast, UCLA and USC (Keck School of Medicine) exemplify academic medical institutions that benefited from good “prenatal” care (endowments), normal birth conditions (well-funded graduate medical education), and developmental maturity (private faculty practice plans and medical facilities). The same can happen in south Los Angeles as was meant to be, and key components of these models need to be embraced and emulated to ensure the mission of Drew is fully realized.

Academic medical centers thrive when they achieve excellence in four critical areas: patient care, teaching, research, and resource generation. KDMC has failed to thrive, because as a two-year medical college, it is continually challenged by poor graduate medical education oversight that is linked to an underfunded county hospital system. It is notable that it is stifled by an antiquated civil service code that is unable to adequately address many of the rapidly changing needs and requirements of an academic medical center. Importantly, these four critical areas are requisite for attracting and retaining the highest quality physician faculty and medical researchers. Moreover, the university suffered maladies in tandem with the hospital’s financial instability and high turnover of leadership. University presidents and deans tend to favor four-year schools and are recognized for their ability to recruit preeminent clinical and academic faculty. This has been a major challenge for KDMC, having had three presidents (one interim) and five deans (three interim) in the past eight years.

However, there is hope on the horizon. The primary goal of the select committee at the King-Drew Medical Center is to restore public confidence in the medical center. Achieving success in this regard will mean correcting the root causes of the problems and a fundamental transformation

beyond a community hospital with residents and faculty. KDMC must move toward a 21st-century model of a successful academic medical center. There has been a lack of and continues to be a need for proactive leadership with innovative ideas that may advance the medical school to a full four-year institution. Leadership who may accrue sustained funding for both the university and the hospital is the only way to enable the provision a full range of quality preventative and interventional health services for a community in dire need.

Each arm of the medical center (university and county) must be held accountable to equally high standards—a changing of the conceptual guard, as it was—that must be realized at the political, structural, and process tiers of medical care in L.A. County. Moreover, this new leadership model must be culturally diverse, knowledgeable, and proactive about crosscultural health issues in order to address the demographic shift that has made the KDMC patient population 65% latino and 30% African American. With this, come more challenges in patient care, physician education, and training and research.

The energy and resolve to address the crisis at KDMC is mirrored in that of two great men: Martin Luther King, Jr. and Charles R. Drew. Their humanitarian efforts gave hope and dignity to the less fortunate and needy in our society. We at Drew and across this great nation who believe in their cause should carry their presence in our hearts and strengthen our resolve to make their dreams a reality. Martin Luther King, Jr. once said, “The greatest form of injustice is inequality in healthcare.” It has also been said, “If you want something you’ve never had, you must be willing to do something you’ve never done.” It’s time to transform the King-Drew Medical Center so that it will thrive to train future generations of physicians who are dedicated to improving the health and well-being of culturally diverse and underserved communities globally.

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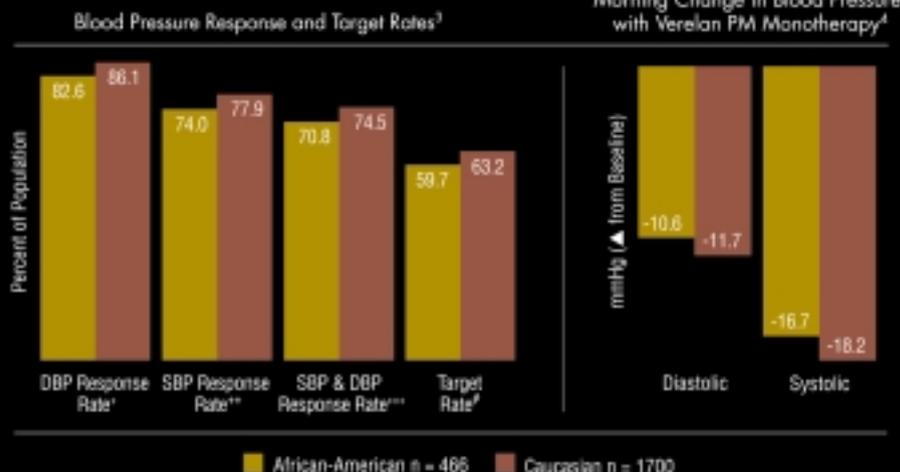
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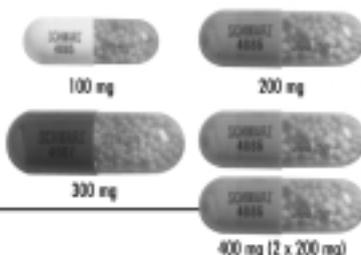
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INDICATIONS AND USAGE: VERELAN[®] PM is indicated for the management of essential hypertension.

CONTRAINDICATIONS: 1. Severe left ventricular (LV) dysfunction (see WARNINGS); 2. hypotension (systolic pressure <90 mm Hg) or cardiogenic shock; 3. sick sinus syndrome (except in patients with a functioning artificial ventricular pacemaker); 4. 2° or 3° atrioventricular (AV) block (except in patients with a functioning artificial ventricular pacemaker); 5. patients with atrial flutter or atrial fibrillation and an accessory bypass tract (e.g., Wolf-Parkinson-White, Lown-Ganong-Levine syndromes; see WARNINGS); and 6. patients with known hypersensitivity to verapamil hydrochloride.

WARNINGS: **Heart failure:** Verapamil has a negative inotropic effect which, in most patients, is compensated by its afterload reduction (decreased systemic vascular resistance) properties without a net impairment of ventricular performance. In previous clinical experience with 4,354 patients primarily with immediate-release verapamil, 1.8% developed congestive heart failure (CHF) or pulmonary edema. Verapamil should be avoided in patients with severe LV dysfunction (e.g., ejection fraction <30% or moderate to severe symptoms of cardiac failure) and in patients with any degree of ventricular dysfunction if they are receiving a β -adrenergic blocker (see Drug Interactions). Patients with mild to moderate LV dysfunction should, if possible, be controlled with optimum doses of digitalis and/or diuretics before verapamil treatment is started (see PRECAUTIONS, Drug Interactions, Digibic). **Hypotension:** Occasionally, the pharmacologic action of verapamil may produce a decrease in blood pressure (BP) below normal levels which may result in dizziness or symptomatic hypotension. The incidence of hypotension observed in 4,354 patients enrolled in clinical trials was 2.5%. In hypertensive patients, decreases in BP below normal are unusual. 70% take timing (BP) was not able to induce orthostatic hypotension. In clinical studies of VERELAN[®] PM, 1.7% of the patients developed significant hypotension. **Elevated liver enzymes:** Elevations of transaminases with and without concomitant elevations in alkaline phosphatase and bilirubin have been reported. Such elevations have sometimes been transient and may disappear even in the face of continued verapamil treatment. Several cases of hepatocellular injury related to verapamil have been proven by challenge; half of these had clinical symptoms (malaise, fever, and/or right upper quadrant pain) in addition to elevations of SGOT, SGPT and alkaline phosphatase. Periodic monitoring of liver function in patients receiving verapamil is therefore prudent. **Accessory bypass tract (Wolf-Parkinson-White or Lown-Ganong-Levine):** Some patients with paroxysmal and/or chronic atrial flutter or atrial fibrillation and a coexisting accessory AV pathway have developed increased atrioventricular conduction across the accessory pathway bypassing the AV node, producing a very rapid ventricular response or ventricular fibrillation after receiving intravenous (IV) verapamil (or digitalis). Although a risk of this occurring with oral verapamil has not been established, such patients receiving oral verapamil may be at risk and its use in these patients is contraindicated (see CONTRAINDICATIONS). **Treatment is usually DC-cardioversion.** Cardioversion has been used safely and effectively after oral verapamil. **AV block:** The effect of verapamil on AV conduction and the SA node may lead to asymptomatic 1° AV block and transient bradycardia, sometimes accompanied by nodal escape rhythms. PR interval prolongation is correlated with verapamil plasma concentrations, especially during the early titration phase of therapy. Higher degrees of AV block, however, were infrequently (0.8%) observed in previous verapamil clinical trials. Marked 1° block or progressive development to 2° or 3° AV block requires a reduction in dosage or, in rare instances, discontinuation of verapamil and institution of appropriate therapy depending upon the clinical situation. **Patients with hypertrophic cardiomyopathy (HCS):** In 125 patients with hypertrophic cardiomyopathy (most of them refractory or intolerant to propranolol) who received therapy with verapamil at doses ≥ 250 mg/d, a variety of serious adverse effects were seen. These patients died in pulmonary edema, all had severe LV outflow obstruction and a history of LV dysfunction. Eight other patients had pulmonary edema and/or severe hypotension; abnormally high (>20 mm Hg) pulmonary capillary wedge pressure and a marked Q2 outflow obstruction were present in most of these patients. Concomitant administration of quinidine (see Drug Interactions) preceded the severe hypotension in 3 of the 8 patients (2 of whom developed pulmonary edema). Since bradycardia occurred in 11% of the patients, 2° AV block in 4% and sinus arrest in 2%. Note that this group of patients had a serious disease with a high mortality rate. Most adverse effects responded well to dose reduction and only rarely did verapamil have to be discontinued.

PRECAUTIONS: THE CONTENTS OF THE VERELAN[®] PM CAPSULE SHOULD NOT BE CRUSHED OR CHEWED.

General: Use in patients with impaired hepatic function: Since verapamil is highly metabolized by the liver, it should be administered cautiously to patients with impaired hepatic function. Severe liver dysfunction prolongs the elimination half-life of immediate-release verapamil to about 14 to 16 h; hence, about 30% of the dose given to patients with normal liver function should be administered to these patients. Careful monitoring for abnormal prolongation of the PR interval or other signs of excessive pharmacologic effects should be carried out. **Use in patients with attenuated (decreased) neuromuscular transmission:** It has been reported that verapamil decreases neuromuscular transmission in patients with Duchenne's muscular dystrophy and it prolongs recovery from the neuromuscular blocking agent vecuronium. It may be necessary to decrease the dosage of vecuronium when it is administered to patients with attenuated neuromuscular transmission. **Use in patients with impaired renal function:** About 70% of an administered dose of verapamil is excreted as metabolites in the urine. Until further data are available, verapamil should be administered cautiously to patients with impaired renal function. These patients should be carefully monitored for abnormal prolongation of the PR interval or other signs of overdosage. **Drug Interactions:** Verapamil undergoes biotransformation by predominantly CYP3A4, however CYP2D6 and members of the CYP2C6 subfamily are involved in its metabolism. Coadministration of verapamil with other drugs metabolized by the above-mentioned enzymes may alter the bioavailability of either verapamil and/or the other drugs. Therefore, coadministration of narrow therapeutic index drugs with similar metabolic pathways as verapamil should be carefully monitored. Similarly, verapamil plasma levels in patients with hepatic dysfunction should be carefully monitored, due to decreased clearance of verapamil in these patients. **Alcohol:** Verapamil has been found to significantly inhibit ethanol elimination resulting in elevated blood alcohol concentrations that may prolong the intoxicating effects of alcohol. **Antihypertensive agents:** Verapamil can increase the efficacy of doxazosin both in tissue culture systems and in patients. It raises the serum doxazosin levels. The absorption of verapamil can be reduced by the cyclophosphamide, anorectic, procarbazine, prednisone (CCPP) and the vindesine, adriamycin, cisplatin (VAC) cytotoxic drug regimens. Concomitant administration of IV verapamil can decrease the clearance of paclitaxel. **Aspirin:** In a few reported cases, coadministration of verapamil with aspirin has led to increased bleeding times greater than observed with aspirin alone. **β -Blockers:** Concomitant therapy with β -adrenergic blockers and verapamil may result in additive negative effects on heart rate, AV conduction, and/or cardiac contractility. The combination of extended-release verapamil and β -adrenergic blocking agents has not been studied. However, there have been reports of excess bradycardia and AV block, including complete heart block, when the combination has been used for the treatment of hypertension. For hypertensive patients, the risk of combined therapy may outweigh the potential benefits. The combination should be used only with caution and close monitoring. Asymptomatic bradycardia (36 beats/min) with a wandering atrial pacemaker has been observed in a patient receiving concomitant timolol (a β -adrenergic blocker) ophthalmics and oral verapamil. A decrease in metoprolol and propranolol clearance has been observed when either drug is administered concomitantly with verapamil. A visible effect has been seen when verapamil and alcohol were given together. **Digibic:** Clinical use of verapamil in digitalized patients has shown the combination to be well tolerated if digoin doses are properly adjusted. However, chronic verapamil treatment can increase serum digoin levels by 50% to 75% during the first week of therapy, and this can result in digitalis toxicity. In patients with hepatic cirrhosis the influence of verapamil on digoin kinetics is magnified. Verapamil may reduce total body clearance and external clearance of digoin by 27% and 29%, respectively. Maintenance and digitalization doses should be reduced when verapamil is administered, and the patient should be reassessed to avoid over- or underdigitalization. Whenever overdigitalization is suspected, the daily dose of digoin should be reduced or temporarily discontinued. On discontinuation of verapamil use, the patient should be reassessed to avoid underdigitalization. In previous clinical trials with other verapamil formulations related to the control of ventricular response in digitalized patients who had atrial fibrillation or atrial flutter, ventricular rate below 50/min at rest occurred in 15% of patients, and asymptomatic hypotension occurred in 5% of patients. **Antiarrhythmic agents:** Verapamil

administered concomitantly with oral antihypertensive agents (e.g., vasodilators, ACE inhibitors, diuretics, β -blockers) will usually have an additive effect on lowering BP. Patients receiving these combinations should be appropriately monitored. Concomitant use of agents that attenuate α -adrenergic function with verapamil may result in reduction in BP that is excessive in some patients. Such an effect was observed in 1 study following the concomitant administration of verapamil and prazosin. **Antiarrhythmic agents:** **Disopyramide:** Until data on possible interactions between verapamil and disopyramide are obtained, disopyramide should not be administered within 48 h before or 24 h after verapamil administration. **Flecainide:** A study in healthy volunteers showed that the concomitant administration of flecainide and verapamil may have additive effects on myocardial contractility, AV conduction, and repolarization. Concomitant therapy with flecainide and verapamil may result in additive negative inotropic effect and prolongation of AV conduction. **Quinidine:** In a small number of patients with hypertrophic cardiomyopathy (HCS), concomitant use of verapamil and quinidine resulted in significant hypotension. Until further data are obtained, combined therapy of verapamil and quinidine in patients with hypertrophic cardiomyopathy should probably be avoided. The electrophysiological effects of quinidine and verapamil on AV conduction were studied in 8 patients. Verapamil significantly counteracted the effects of quinidine on AV conduction. There has been a report of increased quinidine levels during verapamil therapy. **Other: Nitroates:** Verapamil has been given concomitantly with short- and long-acting nitrates without any undesirable drug interactions. The pharmacologic profile of both drugs and clinical experience suggest beneficial interactions. **Cimetidine:** The interaction between cimetidine and chronically administered verapamil has not been studied. Variable results on clearance have been obtained in acute studies of healthy volunteers; clearance of verapamil was either reduced or unchanged. **Lithium:** Increased sensitivity to the effects of lithium (neurotoxicity) has been reported during concomitant verapamil-lithium therapy with either no change or an increase in serum lithium levels. However, the addition of verapamil has also resulted in the lowering of serum lithium levels in patients receiving chronic stable oral lithium. Patients receiving both drugs must be monitored carefully. **Carbamazepine:** Verapamil therapy may increase carbamazepine concentrations during combined therapy. This may produce carbamazepine side effects such as ataxia, headache, ataxia, or dizziness. **Ritanserin:** Therapy with ritanserin may modestly reduce oral verapamil bioavailability. **Phenobarbital:** Phenobarbital therapy may increase verapamil clearance. **Cyclosporin:** Verapamil therapy may increase serum levels of cyclosporin. **Theophylline:** Verapamil may inhibit the clearance and increase the plasma levels of theophylline. **Inhalation anesthetics:** Animal experiments have shown that inhalation anesthetics depress cardiovascular activity by decreasing the inward movement of calcium ions. When used concomitantly, inhalation anesthetics and calcium antagonists, such as verapamil, should each be titrated carefully to avoid excessive cardiovascular depression. **Neuromuscular blocking agents:** Clinical data and animal studies suggest that verapamil may potentiate the activity of neuromuscular blocking agents (curare-like and depolarizing). It may be necessary to decrease the dose of verapamil and/or the dose of the neuromuscular blocking agent when the drugs are used concomitantly. **Cardioversion, Mutagenesis, Impairment of Fertility:** An 8-month toxicity study in rats, at a low multiple (5-fold) of the maximum recommended human dose, not the maximum tolerated dose, did not suggest a mutagenic potential. There was no evidence of a carcinogenic potential of verapamil administered in the diet of rats for 2 y at doses of 10, 35 and 120 mg/kg/d or not 1.3, 4.4 and 15 times, respectively, the maximum recommended human daily dose (400 mg/d or 8 mg/kg/d). Verapamil was not mutagenic in the Ames test in 5 test strains at 1 mg per plate, with or without metabolic activation. Studies in female rats at daily dietary doses <5.9 times (55 mg/kg/d) the maximum recommended human dose did not show impaired fertility. Effects on male fertility have not been determined. **Pregnancy: Category C:** Reproduction studies have been performed in rabbits and rats at oral doses ~ 1.9 (15 mg/kg/d) and 7.5 (60 mg/kg/d) times the human oral daily dose, respectively, and have revealed no evidence of teratogenicity. In the rat, however, this multiple of the human dose was embryotoxic and retarded fetal growth and development, probably because of adverse maternal effects reflected in reduced weight gains of the dams. This oral dose has also been shown to cause hypotension in rats. There are no adequate and well-controlled studies in pregnant women. Animal reproduction studies are not always predictive of human response; this drug should be used during pregnancy only if clearly needed. Verapamil crosses the placental barrier and can be detected in umbilical vein blood at delivery. **Labor and Delivery:** It is not known whether the use of verapamil during labor or delivery has immediate or delayed adverse effects on the fetus, or whether it prolongs the duration of labor or increases the need for forceps delivery or other obstetric intervention. Such adverse experiences have not been reported in the literature, despite a long history of use of verapamil in Europe in the treatment of cardiac side effects of β -adrenergic agonist agents used to treat premature labor. **Nursing Mothers:** Verapamil is excreted in human milk. Because of the potential for adverse reactions in nursing infants from verapamil, nursing should be discontinued while verapamil is administered. **Pediatric Use:** Safety and effectiveness in pediatric patients have not been established. **Geriatric Use:** Clinical studies of VERELAN[®] PM were not adequate to determine if subjects aged 65 or over respond differently from younger patients. Other reported clinical experience has not identified differences in response between the elderly and younger patients; however, greater sensitivity to VERELAN[®] PM by some older individuals cannot be ruled out. Aging may affect the pharmacokinetics of verapamil. Elimination half-life may be prolonged in the elderly. Verapamil is highly metabolized by the liver, and about 70% of the administered dose is excreted as metabolites in the urine. Clinical circumstances, some of which may be more common in the elderly, such as hepatic or renal impairment, should be considered (see PRECAUTIONS, General). In general, lower initial doses of VERELAN[®] PM may be warranted in the elderly. **Animal Pharmacology and/or Animal Toxicology:** In chronic animal toxicology studies verapamil caused ventricular and/or suture line changes ≥ 30 mg/kg/d and tank oysters ≥ 2.5 mg/kg/d in the beagle but not in the rat. Development of cataracts due to verapamil has not been reported in man. **ADVERSE REACTIONS:** Serious adverse reactions are uncommon when verapamil therapy is initiated with upward dose titration within the recommended single and total daily dose. The following reactions to orally administered VERELAN[®] PM occurred at rates $\geq 2.0\%$ or occurred at lower rates but appeared drug-related in clinical trials in hypertension (no. is % in all cases studied): headache (12.1%), infection (12.1%), constipation (8.8%), flu syndrome (5.7), peripheral edema (3.7), dizziness (3.0), pharyngitis (3.0), sinusitis (3.0), dyspepsia (2.7), rhinitis (2.7), diarrhea (2.4), pain (2.4), rash (2.4), asthma (2.0), ECG abnormal (2.0), hypertension (1.7), edema (1.7), nausea (1.7), and accidental injury (1.5). **Infection,** primarily upper respiratory infection and unrelated to study medication. Constipation was typically mild and easily manageable. At the usual once-daily dose of 280 mg, the observed incidence of constipation was 3.9%. **See WARNINGS for discussion of heart failure, hypotension, elevated liver enzymes, AV block, and rapid ventricular response.** Reversible (upon discontinuation of verapamil) non-obstructive, parosmia/leus has been infrequently reported in association with the use of verapamil. In previous experience with other formulations of verapamil, the following reactions occurred at rates $>1.0\%$ or occurred at lower rates but appeared clearly drug-related in clinical trials in 4,354 patients: constipation (7.3%), dizziness (3.3%), nausea (2.7%), hypotension (2.5%), headache (2.2%), edema (1.9%), CHF/pulmonary edema (1.8%), fatigue (1.7%), bradycardia/HR <50 /min (1.4%), rash (1.2%), total AV block, 1°, 2°, 3° (1.2%), 2° and 3° AV block (0.8%), flushing (0.6%) and elevated liver enzymes (see WARNINGS). In clinical trials related to the control of ventricular response in digitalized patients who had atrial fibrillation or atrial flutter, ventricular rate below 50/min at rest occurred in 15% of patients and asymptomatic hypotension occurred in 5% of patients. The following reactions, reported with orally administered verapamil in $\geq 2.0\%$ of patients, occurred under conditions where a causal relationship is uncertain: angina pectoris, atrioventricular dissociation, chest pain, claudication, myocardial infarction, palpitations, pupura (vasculitis), syncope, diarrhea, dry mouth, gastrointestinal distress, gingival hyperplasia, ecchymosis, bruising, cerebrovascular accident, confusion, equine distemper, ischemia, muscle cramps, paresthesia, psychotic symptoms, shakiness, somnolence, depression, ataxia and rash, anaphylaxis, hair loss, hyperkalemia, macules, swelling, urticaria, Stevens-Johnson syndrome, erythema multiforme, blurred vision, linitis, gynecoma, galactorrhea/hyperprolactinemia, impotence, increased urinalysis, spotty menstruation, and allergy aggravated. **Treatment of Acute Cardiovascular Adverse Reactions:** Cardiovascular adverse reactions rarely require therapy; hence, treatment experience is limited. When severe hypotension or complete AV block follows oral administration of verapamil, appropriate emergency measures should be applied immediately, e.g., IV-administered norepinephrine bitartrate, atropine sulfate, isoproterenol HCl (in usual doses), or calcium gluconate (10% solution). In patients with hypertrophic cardiomyopathy (HCS), α -adrenergic agents (phenylephrine HCl, metaxalone bitartrate, or methoxamine HCl) should be used to maintain BP, and isoproterenol and norepinephrine should be avoided. If further support is necessary, dopamine HCl or dobutamine HCl may be administered. Actual treatment and dosage should depend on the severity of the clinical situation and the judgment and experience of the treating physician.

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Recruitment in Social and Behavioral Research National Human Genome Research Institute • National Institutes of Health

The Social and Behavioral Research Branch (SBRB) is a newly-formed Branch of the National Human Genome Research Institute whose mission is to conduct innovative research in:

- applying genetic discoveries to improve interventions for disease prevention and health promotion,
- evaluating genetic risk communications,
- developing for communicating genetic risk to affected individuals, families, communities, and populations,
- understanding how social factors influence genetic discoveries and research, and
- investigating the ethical and public policy implications of genetic research and the use of genetics in clinical practice.

The SBRB currently consists of seven investigators whose research is at the intersection of genomics and social and behavioral science, including clinical genetic counseling, risk communication, health behavior change, bioethics and social policy, health disparities, community research, and public health.

We are seeking dynamic investigators at various levels (staff scientists, tenure track and tenured) to join us in meeting our goal of becoming one of the premier programs in this area. New SBRB investigators will be expected to develop an independent research program in keeping with the Branch's broad research mission. Candidates should have expertise or interest in conducting innovative research on behavioral and social aspects of human genome discoveries. Prior genomic research experience is desired but not required. General areas of interest being targeted for recruitment include but are not limited to:

- Risk communications
- Behavior change interventions
- Decision assistance interventions
- Health services research
- Community involvement research
- Health economics
- Mass communications
- Bioethics
- Public policy and regulation
- Research protections
- Health Disparities

Candidates must have a Ph.D., M.D. or equivalent degree. Rank will be commensurate with qualifications. The positions include an ongoing commitment of research support and space, support personnel, and post-doctoral positions.

Interested applicants should send a curriculum vitae, a three-page description of research interest and vision, and three letters of recommendation through our online application system at <http://research.nhgri.nih.gov/apply>. Applicants who cannot submit their materials electronically should submit their applications to:

Monique White
SBRB Search Committee
National Human Genome Research Institute
50 South Drive
Building 50, Room 5349
Bethesda, MD 20892-8000
mwhite1@mail.nih.gov

Applications will be reviewed beginning February 15, 2004 and continue until positions are filled or September 30, 2004. For more information on SBRB and NHGRI's Intramural Program, <http://www.genome.gov>



Fellowships Leading to a Masters of Science in Clinical Investigation

Meharry Medical College, located in Nashville, TN, is offering two-year fellowships for Clinical Research and Education for Career Development (CRECD) leading to a Masters of Science in Clinical Investigation (MSCI) degree. The program will expand the cadre of well-trained researchers, particularly minorities, to foster careers in clinical investigation concentrating on health disparities. The curriculum includes core didactic, elective and experimental design sessions that span molecular medicine, clinical research, epidemiology and health services research. Courses newly created for this program focus on health disparities, culture and health, clinical research methodology and research ethics. Courses are provided both at Meharry Medical College and Vanderbilt University and include small group seminars for interaction and discussions between trainees and faculty. Each trainee, during the first year, will establish a mentoring committee composed of faculty from both Meharry and Vanderbilt and create a research project. The mentoring committee will review the project and assist the trainee in design, conduct, implementation and analysis. The program offers tuition, book allowances, supplies and salary support along with resources to conduct the clinical trial. Applicants must be U.S. citizens, non-citizen nationals or lawfully admitted permanent residents of the U.S. For program inquiry contact: Steven N. Wolff, M.D., Meharry CRECD/MSCI Program at phone (615) 327-6763 or e-mail swolff@mmc.edu.



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- Division of Nephrology
- Division of Pulmonary and Critical Care
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Interested applicants may learn more by viewing our website at www.musc.edu or may forward a CV to glanvilf@mus.edu or to Frances Glanville, Department of Medicine, 96 Jonathan Lucas Street, PO Box 250623, Charleston, SC 29425.